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Surgical Treatment of Chronic Constrictive Pericarditis

A CLINICAL STUDY OF 38 CASES WITH SPECIAL

REFERENCE TO THE VALUE OF THE LEFT

TRANSPLEURAL APPROACH

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FROM THE DEPARTMENTS OF SURGERY, SABBATSBERGS SJUKHUS AND THE
THORACIC CLINICS (SURGEON-IN-CHIEF: PROFESSOR CLARENCE CRAFOORD),
KAROLINSKA SJUKHUSET, STOCKHOLM, SWEDEN.

Surgical Treatment of Chronic Constrictive Pericarditis

A Clinical Study of 38 Cases, with Special

Reference to the Value of the Left

Transpleural Approach

By

LENNART JOHANSSON

STOCKHOLM 1958

Translated from the Swedish by

ERICA ODELBERG

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PREFACE

Professor Clarence Crafoord's pioneer work in heart and lung surgery is so widely recognized that any recapitulation here would be superfluous. His stimulating and unfailing interest in the present investigation has greatly facilitated its performance, and it is a pleasure to acknowledge my deep debt of gratitude to him.

I take this opportunity of expressing my sincere thanks to Professor Hilding Berglund and his co-workers at S:t Eriks Sjukhus, as well as to Professor Gustav Nylin and his co-workers at Södersjukhuset, for their kindness in placing at my disposal the case records and other data of patients referred for operation.

The cardiac catheterizations were performed at the Cardiorespiratory Laboratory of Sabbatsbergs Sjukhus, whose chief, Docent Sven Åkesson, kindly allowed me to make use of the resources of his laboratory. In the course of the investigations, I received much valuable assistance from Drs. Marianne Bagger and Hans Erik Hansson, Docent Gunnar Malmström and Dr. Lars-Gustaf Uggla.

Some of the roentgenologic examinations were performed at Sabbatsbergs Sjukhus, where Docent Bengt Holmgren gave me the benefit of his valuable collaboration. Part of the angiocardiographic investigations were made at Södersjukhuset, where Docent Gunnar Jönsson and his co-workers placed their skilful assistance at my disposal. A great number of the angiocardiographies, as well as the electrokymographic examinations, were carried out at the Roentgendiagnostic Department of the Paediatric Clinic, Karolinska Sjukhuset, where the great experience and technical knowledge of Docent Sven Roland Kjellberg and Docent Ulf Rudhe were an invaluable asset. I am also greatly indebted to Docent Ulf Rudhe for many stimulating discussions regarding the electrokymograms.

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The manuscript was translated from the Swedish by Mrs Erica Odelberg.

I offer my sincere thanks to all of them for their assistance.

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Stockholm, March 1958

LENNART JOHANSSON

INTRODUCTION

Both formerly and in recent years, stenosis of the venae cavae and damming up of the blood have often been suggested as an explanation of the symptoms of congestion in chronic constrictive pericarditis. If this were actually the case, it should be possible to demonstrate such patho-anatomic changes not only at operation, but also before it, by depiction of the contrast-filled caval veins. After the introduction of angiocardiology at Sabbatsbergs Sjukhus about 1946, the true anatomic conditions could be established by this means.

On the basis of earlier experience, confirmed by the results of angiocardiological examinations, we gradually formed the opinion that operation for chronic constrictive pericarditis should be performed at two sessions. At the first session, decortication of the ventricular region was to be carried out through the left transpleural approach. Since it was considered that any involvement of the venae cavae could scarcely be of importance as an impediment to inflow, they could be decorticated, if necessary, at a second session. With few exceptions, this opinion has been decisive for the surgical technique.

Although the results of operation were striking in most cases, it was considered that a more detailed analysis and evaluation of them were imperative for guidance in the future. The present investigation, based on a series of 38 operated cases, has been made with this purpose in mind.

CASE MATERIAL

The present investigation is based on a series of 38 cases of chronic constrictive pericarditis, operated on during the 1944 to 1955 period. The presence of chronic constrictive pericarditis could be confirmed at operation in every case. Thirty-seven patients underwent one operation, whereas two operations were performed in the remaining case.

PART ONE: HISTORICAL

CHAPTER 1

REVIEW OF EARLIER INVESTIGATIONS

EARLY OBSERVATIONS OF PERICARDIAL DISEASE

RICHARD LOWER was the first to give a reliable account of pericardial disease in man. In 1669, he described the physiopathologic features of exudative pericarditis, and the course of events in adhesion of the pericardium to the heart and diaphragm. Thus, he initiated the era of recognition of acute and chronic pericarditis some years before cases were mentioned by BONNETUS (1679) and by VIEUSSENS (1715). RICHARD LOWER wrote:

"While the heart's capsule is stated to be of service to it on more than one count, serving indeed, both for its moistening and for its protection from outside harms, it is also often injurious to the heart in more than one way. For just as it injures the heart by accumulation of fluid within it, so, when this is completely absent, it approaches so close to the heart, that at length it adheres everywhere to this organ. Hence, as it is also joined to the diaphragm, it must combine and unite the heart's movement with that of the diaphragm. How great a hindrance and disadvantage this must be for both organs I have shown above, and it will be still clearer from the following story.

The wife of a certain citizen of London, aged 30, healthy and active enough previously, became very dejected and melancholy during the last three years of her life, suffered from breathlessness on the least exertion, had a small and often an intermittent pulse, and complained almost continuously of attacks of pain and of great physical discomfort in the precordium. She had at last become subject to frequent fainting fits, and to loss of consciousness and chilling of the extremities on the gentlest movement of the body. No treatment was of any avail to her in this condition, and, her strength at length slowly exhausted, she died. When the body was opened, no abnormalities at all were visible among the abdominal viscera. While examining the other organs, however, we discovered a pathological condition of the heart, to which we may rightly attribute the cause of all her troubles. The thorax was opened and the lungs were healthy enough, the pericardium, however, had become closely attached all over the whole surface of the heart, so that it could only with difficulty be separated from it. Further, this membrane had become thick, opaque and hard instead of being thin and transparent, as it should naturally have been. Hence, as there was no space for the free movement of the heart, and no fluid for moistening its surface, it is little wonder that she complained all the time of these ills.

Further, as the diaphragm is always attached to the pericardium in man, when the heart itself was also united to the pericardium, the diaphragm must of necessity have carried the heart down with it at every inspiration, and during that time must have held up and suppressed its movement. So the observed intermission of the pulse succeeded regularly at every inspiration."

It is apparent that LOWER was the first to detect the paradoxical pulse. His observation nevertheless fell into oblivion until 1873, when the phenomenon was rediscovered by KUSSMAUL, who since then has erroneously been regarded as its discoverer.

In the 18th century, VIEUSSENS (1715) and BOERHAVE (1768) suggested that adherent pericardium could be a cause of heart disease. In 1729, LANCISI described a 24-year-old man with a small pulse, swelling of the neck veins and a swollen abdomen, autopsy showing a small heart surrounded by thick, adherent pericardium.

In 1761, MORGAGNI gave an account of seven cases of the disease in which the heart was so constricted that it was unable to receive sufficient blood to supply the body. He pointed out that the myocardium could also be involved, and thus undergo calcification, and that a weakened or absent apex impulse was an important symptom. He stated, however, that most patients were little or not at all disabled by the disease. P. D. WHITE (1935), who cited MORGAGNI, expressed the opinion that this observation was responsible for interest in the disease being lost for a long time.

LIEUTAUD (1767) described 23 cases in which the symptoms were dyspnoea, a small pulse, oedema and ascites.

In 1818, CORVISART reported three cases in which death occurred 12 months after the onset of oedema and ascites. The earliest reports attached particular importance to the small, quiet heart and the clinical features of oedema and ascites. HOPE, in 1832, was the first to mention that adherent pericardium could produce enlargement of the heart. The subsequent description by BROADBENT (1895) laid such stress on the enlarged heart that this came to be accepted as the most important and only effect of the disease. NORMAN CHEVERS (1842) had nevertheless emphasized that he had never observed cardiac enlargement in adhesive pericarditis unless there was coincident valvular disease. He wrote: (The fact that) "... in nearly half of the cases of partial adhesions the heart retained its ordinary dimensions, while in the remainder there was sufficient valvular disease to account for any enlargement that was present, would tend to prove that partial adhesion of the pericardial surfaces is not more liable to produce hypertrophy with dilatation than

is the more complete form of the disease." According to Chevers, pericardial fibrosis had a tendency to produce a generalized decrease in the size of the heart and its vessels, a contraction of its chambers, and an impediment to both systole and diastole, particularly the latter.

In *The Harmlessness of Adherent Pericardium*, KING (1845) analyzed two autopsy series, on one of which Chevers' view was based. He came to the conclusion that pericarditis did not produce any great degree of cardiac enlargement, but that if this occurred it was due to valvular disease, for which the endocarditis coincident with pericarditis was responsible.

OBSERVATIONS OF CHANGES IN OTHER ORGANS

Up to this date, the main interest had been focused on the cardiac lesions, and little or no study had been made of the changes in other organs. Thus RICHARD LOWER, for example, had stated — in his aforementioned autopsy report — that no pathologic changes were present in the abdomen. It is true that earlier workers had observed cases in which ascites was present, oedema was not conspicuous, and a diagnosis of cirrhosis of the liver had been made, pericarditis being disclosed only at autopsy. These cases had not, however, aroused much attention. The earliest case mentioned was that of VAN DEEN (1846). Cases were later described by many authors (BROADBENT; DICKINSON; FEIERABEND; FRIEDREICH; HAMBURGIN; HARRIS; HENOCHE; HIRSCHLER; MOTT; RIEDEL; TISSIER; VARIOT; VIERORDT; WEINBERG; WEISS). Chronic hyperplastic perihepatitis — frosted liver or Zuckergussleber — was described by ROKITANSKY (1842), BAMBERGER (1864), MURCHISON (1868), FAGGE (1875), MOXON & WILKS (1875), and CURSCHMANN (1884). In 1887, ROSENBACH noticed that pericardial obliteration was sometimes associated with fibrous perihepatitis, enlargement of the liver and ascites. He concluded that there was some relation between these features and adhesion of the pericardium, a fact also stressed by STRÜMPPELL (1890) and LEUBE (1891). SCHRÖTTER stated in 1894 that pericardial obliteration sometimes gave rise to ascites before the appearance of generalized anasarca.

In 1896 and 1899, FRIEDEL PICK pointed out that the recurrent ascites, with an almost complete lack of oedema of the legs, the enlarged, firm and somewhat irregularly shaped liver, with no or only inappreci-

able jaundice, indicated the presence of cirrhosis of the liver. Moreover, obliterating pericarditis usually escaped clinical detection, even if he had been able to establish the diagnosis during life in one of his patients. PICK suggested the term *pericarditic pseudocirrhosis of the liver* for this condition, and regarded pericarditis as the primary cause of the circulatory disturbance. He interpreted the cases described earlier by CURSCHMANN and by RUMPF as pseudocirrhosis on a pericarditic basis.

PICK's publications gave rise to a lively discussion. Some authors (*e. g.* BOZZOLO; GALVAGNI; NACHOD; PATELLA and SIEGERT) agreed with PICK, and considered ascites to be a result of the liver stasis produced by pericarditis. Others, on the contrary (*e. g.* HEIDEMANN; SCHUPFER and WERBATUS) regarded the chronic peritonitis observed at autopsy to be responsible for ascites. HARRIS and OSLER, among others, attached the main importance to these two causes. WEINBERG explained the ascites as an angulation of the inferior vena cava and the hepatic veins, owing to the right-sided pleural exudate. This statement was based on some experiments of ROSENBAACH, which seemed to point in the same direction. Pick emphasized that the old rule that oedema of the legs predominates in heart disease, and ascites in liver and portal disease, was not fully correct. This was of practical importance, since in these cases treatment of cirrhosis was ineffective, whereas benefit was derived from treatment of the heart condition.

SYMPTOMATOLOGY

As far as the symptomatology is concerned, LOWER had already described the paradoxical pulse, which was rediscovered and named by KUSSMAUL in 1873. KUSSMAUL explained the respiratory variation in amplitude as a result of adhesions round the great vessels, which became mechanically kinked or displaced during the respiratory excursions. Contrary to the normal conditions, deep inspiration produced swelling of the neck veins, especially of the bulb of the jugular vein. When all other findings supported the diagnosis of obliteration of the pericardial cavity, this phenomenon was considered to provide an additional degree of certainty. KUSSMAUL recalled that it was described for the first time by GRIESINGER (1854) and by WIDENMANN (1856).

These symptoms and signs were complemented by others regarded to indicate adhesion of the heart to the adjacent structures. CORVISART

pointed out a feeling of traction in the heart region, MORGAGNI absence of the apical impulse, and HEIM systolic retraction in the epigastric angle. Distinct awareness of the heart action was mentioned by WILLIAMS, systolic retraction at the site of the apical impulse by SKODA, and disappearance of the second heart sound by ARAN. FRIEDREICH pointed out the systolic retraction of the anterior thoracic wall, ribs and sternum, and BROADBENT systolic retraction between the eleventh and twelfth ribs to the left of the spinal column. Finally, BRAUER and FRIEDREICH described diastolic shock, which could sometimes be heard with the stethoscope as a third heart sound.

INTRODUCTION OF SURGICAL TREATMENT

Consequently, it was natural that once the morbid anatomic aspects and the clinical features had been combined into a clinical entity — even if it was the subject of no little controversy — the question of treatment would not be far distant. It was chiefly two European workers who studied the possibilities of treating the disease. WEILL (1895) was presumably the first to suggest freeing of the adhesions in the pathologic pericardium. DÉLORME may concurrently have observed at the autopsy table that it was not difficult to free the heart from the adherent pericardium. He did not, however, publish his observations until 1898, but since then cardiolysis has often been known as DÉLORME's operation.

DÉLORME suggested that a 7 to 8 cm long incision be made at the level of the fifth rib, with removal, if necessary, of parts of the cartilage of the third and fourth ribs. The pericardium should be split horizontally, to allow access to the lateral parts. A finger and blunt instruments were used for freeing the adhesions. The author recommended that the operation be performed on young subjects, and on an inappreciably dilated heart, that had not reached the borderline of degeneration or heart failure. DÉLORME wrote:

"... il consiste dans la section ou la destruction des adhérences cardio-péricardiques: c'est appliqué au coeur, l'acte opératoire que j'ai imaginé pour le poumon. Cette symphyse ne trouble pas seulement les mouvements de systole et de diastole du coeur, elle limite ou empêche sa locomotion systolique en bas et en avant et son mouvement de torsion."

He also gives advice on the conduct of the surgeon.

"Est-il nécessaire d'ajouter que l'opérateur doit ici se faire une règle d'une prudence qui n'exclut pas la hardiesse, qu'il doit toujours voir ce qu'il fait, se rapprocher du péricarde pariétal et procéder lentement."

In America, the question was probably raised for the first time by BECK in 1901. In a contribution to a discussion of pericarditis, he wondered whether the adhesions could not be attacked surgically.

In 1902, BRAUER reported a method for surgical treatment of adhesive mediastino-pericarditis. He distinguished between internal adhesions, which lead to obliteration of the pericardial cavity, and external adhesions, which bind the heart to the intrathoracic structures. He stated the latter to be the more important, since the heart is unable for any length of time to perform the extra work implied by retraction of the bony parts of the thoracic wall during systole. In cases with marked systolic retraction, BRAUER recommended resection of the bony part of the thoracic wall covering the heart, so that it was covered by resilient tissues. In 1903, he gave an account of his experience of this operation, and demonstrated a case in which ascites had disappeared postoperatively. The technique used in BRAUER's first operations was elaborated by MEYER-WESTFELDT, by VON BECK and by DANIELSEN.

BLAUER (1907) reported three cases and KÖNIG (1907) one case in which benefit had been derived from cardiolysis according to BRAUER.

In England, BRAUER's operation was performed for the first time by WENCKEBACK in 1907. Together with a report of a case, he published some reflections on the disease. He stated that, owing to fixation of the heart to its surroundings, pericardial adhesions prevent not only a normal heart action, but also normal respiration. WENCKEBACK assumed that, instead of the normal expansion of the chest in both diameters, the thorax is retracted by the adhesions around the heart, and descent of the diaphragm is impeded. This, as well as contraction during respiration of the diaphragmatic crura enclosing the inferior vena cava, prevents emptying of the blood from the splanchnic region, and leads to enlargement of the liver and ascites. In the patient in question, enlargement of the liver persisted after operation, which was nevertheless regarded as successful.

In America, SUMMERS was the first, in 1912, to perform BRAUER's operation.

Among the earliest operations for removal of the constricted pericardium according to DÉLORME, were those of HALLOPEAU (1910), which subsequently fell into oblivion, and of REHN (1912) and SAUERBRUCH (1913). A consecutive series was reported more than 10 years later by VOLHARD & SCHMIEDEN.

In 1914, DÉLORME published a further account of his experience of

“symphyse cardio-péricardique”. He recommended early operation in both children and adults. He stressed that in the suppurative form it is necessary to wait until the symptoms have abated or disappeared, but also that decortication is a complement to pericardiotomy in this form of the disease. He stated that operation should be performed in rheumatic and tuberculous pericarditis, and even when the heart is hypertrophied or dilated, although greater caution is required in the last-mentioned cases. DÉLORME had noted that a layer of the visceral pericardium sometimes remained after decortication, but he did not believe that this could ever be removed without serious complications. He was of the opinion that it was as risky to incise this layer as to remove it.

As early as the end of the 19th century, BARNARD and others had studied pericardial function. Experiments in this field were continued by REHN, for example. Attempts were made to elucidate the pathophysiology of constrictive pericarditis, and on the basis of these investigations to work out the best methods of operation.

In 1920, REHN stated that the pericardial changes primarily affect the contractility of the thin-walled atria, having less effect on that of the ventricles, especially the left. The inadequate atrial contraction causes blood to remain in the right atrium, with decreased filling of the ventricle as a result. The atrium becomes dilated, and its contraction produces reflux to the veins, since they are incompletely shut off. REHN based these statements on TANDLER & KEITH's studies of occlusion of the sinus region during atrial systole. He described the subsequent events as follows. The reflux to the veins leads to stasis. Involvement of the right atrium is followed by that of the thin-walled right ventricle, which reacts similarly by over-filling during diastole. Later, when the left atrium also becomes incompetent, pulmonary stasis develops, since the pulmonary veins have a kind of sphincter mechanism. The leading symptom is stasis in the region of both caval veins. Enlargement of the liver is ascribed to the thin veins, devoid of valves, which open into the inferior vena cava at right angles to it. With successive shrinking of the adhesions, diastole is also impeded, and the heart finally becomes walled-in and bound, and the whole thorax in the heart region becomes immobile. REHN stressed that this is of particular consequence in childhood, when the heart is more thin-walled; it continues to grow, and the space available in the thorax is then of some importance.

VOLHARD introduced the term “Einflußstauung” for the main symptom, *i. e.*, stasis in the systemic veins.

Operative treatment of the disease had been taken up by SCHMIEDEN in 1918, and in 1923 he published, together with VOLHARD, an account of his experience hitherto. VOLHARD & SCHMIEDEN recalled the observation made by CHEVERS as early as 1842, namely, that the diseased pericardium forms an impediment to both systole and diastole, but chiefly the latter. They stated that simple adhesions do not necessarily produce any symptoms, but that fibrous thickening and shrinking of the pericardium lead to the typical symptoms. They distinguished between two types, *accretio cordis*, in which the heart is bound to adjacent structures, and *concretio cordis*, in which there is adhesion of the visceral and parietal pericardium. In their experience, however, it was rare to find either type isolated, a combination of both types usually being present. They stressed that treatment should be surgical, and should consist of removal of the fibrous pericardium encasing the heart.

The authors pointed out that, in decortication, it is important to free the ventricles, particularly the left, on which they considered the whole circulation to depend. Decortication of the right ventricle was regarded as harmful, and could lead to its overdistension, unless the left ventricle was freed concurrently. This was stressed by SCHMIEDEN, who was responsible for the surgical aspects; he stated that right-sided decortication had resulted in death in one of his cases. He pointed out that the pericardium protects the right ventricle from acute overdistension and tricuspid incompetence. The object is to free the functionally most important part of the heart, this being the ventricles. But it is also necessary to debate which part should not be decorticated, since complete decortication is tolerated only by a heart with exceedingly strong muscles.

SCHMIEDEN emphasized the large part of the pericardium that is adherent to the diaphragm, and the disturbing influence of the latter on inspiration. In such cases, he suggested division of the phrenic nerve, which implied that this nerve was not freed before decortication of the left ventricle, but was sacrificed on removal of the pericardium. SCHMIEDEN also favoured left-sided decortication because he considered that it might be difficult to find a suitable layer in which to work, and there is far less risk of injury to the strongly muscled left ventricle than to the right. He ascribed to the right atrium an important nervous influence on the heart action, and definitely advised against any intervention on this chamber. SCHMIEDEN was the originator of the well-known comparison of decortication to the peeling of an orange. SCHMIEDEN treated seven

patients, one of whom was operated on twice; three recovered and two died in connexion with operation.

Brauer's operation had, however, been performed for a longer time than decortication according to DÉLORME, and a larger number of cases had therefore been collected. A series was reported by BOURNE in 1924 and another by SCHLOFFER in 1929. None of BOURNE's cases seemed to be convincingly improved by operation alone. SCHLOFFER was entirely in agreement with BRAUER in his report of 60 cases with one death. He stated that this operation was definitely indicated when systolic retraction was present as a sign of extrapericardial adhesions. He expressed no precise views on decortication according to DÉLORME, but stated that the cases in which it is suitable are more difficult to find, owing to the more indefinite symptoms; consequently, this difficult operation was likely to be performed only infrequently. No definite conclusions can be drawn regarding the results in the 107 cases of decortication reported by SMITH & LIGGETT (1928), owing to the classification of the material.

Although BECK had been engaged in experimental investigations of pericardial disease since the early 1920's, decortication according to DÉLORME was not performed in America until 1928 by CHURCHILL. In 1929, he made some observations in connexion with the operated case. He pointed out both the extrapericardial adhesions which impede the heart in systole, particularly since the myocardial changes decrease the possibility of carrying out this extra work, and the thickened, inelastic pericardium, which encases the heart in an envelope, and impedes diastolic filling. In the former case he recommended BRAUER's operation, but in the latter he regarded decortication as the only correct method. He considered it unfortunate that no reliable criterion of the capacity of the myocardium existed, so that it could be decided whether the heart would tolerate the stress resulting from decortication. According to CHURCHILL, it might be highly detrimental for the output of the right side of the heart to be increased by decortication, as long as the left ventricle remained embedded in scar tissue. From the literature up to 1929, he collected 42 cases, with a postoperative mortality of 7 out of the 37 cases in which operation was completed, and death was directly attributable to the intervention. Of the remaining 35 patients, 19 had benefited by operation, and were virtually asymptomatic. Churchill added a case of his own in which decortication was performed, and was followed by complete recovery.

INVESTIGATION OF PHYSIOLOGIC ASPECTS

Under the impression that decortication according to DÉLORME was a practicable form of treatment in chronic constrictive pericarditis, more detailed clinical analyses of the physiologic aspects were started in the 1930's. Surgical treatment had been suggested and introduced by European workers; the physiologic investigations were continued by workers in America.

The diseased pericardium was considered to form the circulatory impediment, whereas the extracardiac adhesions were of no consequence. BURWELL *et al.* (1932) stated that the thickened pericardium not only impedes diastolic filling, but must also make systolic contraction more difficult. They wrote:

"It seems doubtful in many instances that the heart could expel the normal quantity of blood at each contraction even if it could receive it. The inextensible and relatively unyielding scar which is attached to the entire surface of the heart must offer a real impediment to systolic contraction. Shortening and rotation of the heart in systole are almost entirely abolished."

Obstruction of emptying of the venae cavae into the heart nevertheless continued to be counted as a factor responsible for the circulatory changes. It was mentioned by SPRAGUE & WHITE (1932) and by SCHUR (1934), whereas SCHMIEDEN & WESTERMANN (1937) stressed that the pericardium was the obstacle both to emptying of the caval veins and to ventricular action. It was stated that the pulmonary artery could also be constricted by the adhesions (GOULEY 1937). The inferior vena cava was considered to be more liable to constriction by the pericardium. In order to rule out this possibility, BURWELL and his co-workers studied the pressure in the region of both caval veins. They found the difference to be so inappreciable that there was no reason to suspect any difference between the obstruction of the superior and the inferior vena cava. They observed that the venous pressure rose steadily during the whole time preceding operation. The blood volume was larger than normally, and the circulation time was prolonged, an observation confirmed by GIBSON & EVANS (1937).

BURWELL *et al.* observed that spontaneous or induced changes in blood volume produced parallel changes in venous pressure, whereas stroke volume, heart rate and circulation time were unaffected. They found, as did MALTBY (1934) and STEWART and his co-workers (1938, 1939), a

low stroke volume and raised arteriovenous oxygen difference. The low stroke volume led to tachycardia, low pulse pressure and limitation of the working capacity. The increase in venous pressure was the chief factor responsible for such symptoms and signs as oedema, ascites and enlargement of the liver.

In 1937, BURWELL summarized his views at a meeting of the American Heart Association as follows:

"This question is concerned with the place in the circulation at which the obstruction occurs. When the pericardium is filled with fluid, it is obvious that the compression of the heart is exerted generally. It must involve the ventricles, the auricles and the intra-pericardial portions of the great vessels. One sometimes gets the impression from reading the older literature that the obstruction from scar comes about through constriction from one of the vena cava. If constriction of the venous channels were important, one would expect to find occasionally a difference in the pressure in the inferior vena cava as compared with the superior vena cava system. In a considerable number of cases in which comparable measurements have been made, we have not seen a case in which such a difference existed, except when some local cause for it was present.

If I understand DR. BECK's description, it implies that he considers the important point of obstruction to be in the pericardial scar involving the ventricles themselves. This agrees with the general concept that the difficulty in constrictive pericarditis is the inability of the ventricle to dilate adequately. If this theoretical concept is true it should be demonstrable by a simple experiment. If the heart cannot dilate and its output per beat is fixed at a low level, the only way the cardiac output per minute can increase is by acceleration of the pulse rate. This being so, if the pulse rate be accelerated for example by atropine, one might expect the venous pressure to be reduced. This is in fact the case. When, by the use of atropine, the heart rate of a patient with constrictive pericarditis was elevated from 80 per minute to 120 per minute, the venous pressure fell to a significant degree."

According to SCHMIEDEN & WESTERMANN (1937), the clinical features are the same as those of mitral stenosis, and it is difficult to make a differential diagnosis between these two conditions. WHITE (1935) as well stressed the similarity to mitral stenosis, but also stated that the symptoms persisting after operation are due to constriction of the left ventricle, particularly of the atrioventricular border, and that this could produce a picture resembling that of mitral stenosis. BECK (1935) denied any such influence of the pericardium, and described the features of acute and chronic compression in his two "compression triads". In chronic compression, they were stated to consist of high venous pressure, ascites and a small, quiet heart.

INFLUENCE OF PHYSIOLOGIC ASPECTS ON SURGICAL TECHNIQUE

It was unanimously agreed that operation should be performed at the optimal time, but since many regarded tuberculosis to be one of the chief causes of constrictive pericarditis, it was considered advisable to wait until this was no longer in the active stage. CHURCHILL (1936) had found the results of operation during the active stage to be extremely disappointing. However, since symptoms of compression did not invariably appear in the inactive phase of tuberculosis, and *vice versa*, BLALOCK & LEVY (1937) were of the opinion that if the symptoms of stasis could not be kept under control by conservative measures, operation was obligatory despite the active stage.

Opinions were highly divergent regarding the factor chiefly responsible for the circulatory disturbance. Consequently, it was natural for the views on the best way of dealing with the compression to be equally variable. Some surgeons considered that the results of operation depended on whether the caval veins were decorticated (BECK & GRISWOLD 1930, TENG-VALL 1938). WINKELBAUER & SCHUR (1936) stated that the caval veins should be decorticated if ascites was conspicuous; if, on the contrary, hydrothorax was present, the left ventricle should be decorticated. It was considered dangerous to intervene on the thin-walled atria, although BECK (1931) maintained that the pericardium must be removed from them. SCHMIEDEN & WESTERMANN and CHURCHILL stressed that it was difficult to decorticate the atria without injury to them, and that the pericardium should therefore be left at this site, and decortication interrupted at the atrioventricular groove, CHURCHILL pointing out that it was important to free the right atrioventricular border.

That decortication is not as easy as would appear from SCHMIEDEN's comparison to the peeling of an orange was shown by the observations of CHURCHILL (1936) and BLALOCK & LEVY (1937) that the coronary vessels are sometimes more adherent to the scar than to the heart, and that the pericardial scar should be resected as close to the myocardium as possible. The difficulty of removing the pathologic tissue was illustrated by FLICK & GIBBON (1934), who reported a case in which operation led, not to improvement, but to death. At autopsy, no stenosis of the venae cavae was found, but the epicardium still covered the heart and was partly calcified.

Intervention was not confined to the constricted scar tissue. The

phrenic nerve was sacrificed in cases in which, owing to adhesions, the action of the heart and diaphragm were in opposition (TROUT 1931).

BECK's statements, made in 1937, regarding constrictive pericarditis summarize the conception that started to be prevalent at this time. He stated that the compressed heart is a small, quiet organ. It is prevented from receiving its normal quantity of blood, so that the cardiac output is reduced and it undergoes atrophy of disuse, in similarity to other organs that are not used. The heart functions effectively but is not allowed to function adequately. Involvement of the liver and spleen is due, not to infection, but to venous stasis. Adhesion of the heart to adjacent structures produces no symptoms unless there is rotation or angulation of the heart. BECK was of the opinion that, if hypertrophy supervened, it was to be ascribed to valvular disease. He also rejected the value of cardioly-sis according to BRAUER in the following words:

"If I might express an opinion, I would say that the extra bed rest and attention afforded these patients before and after operation did them more good than the operation itself. Even this statement implies that the operation did some good and of this I am sceptical."

In an analysis of adherent pericardium, ARMSTRONG (1940) and SODEMAN (1941) distinguished between cases with and without compression. Histologic examination clearly showed which tissue was responsible for the constriction. They expressed the view that this difference in appearance also indicated a difference in aetiology.

BURWELL still maintained that the cause of the circulatory disturbance lay, not in constriction of the large afferent vessels, but in the inability of the heart to become adequately dilated. The mediastino-pericarditis leading to extrapericardial adhesions was ascribed to rheumatic infection, and the cardiac enlargement to valvular disease rather than to the adhesions.

Many authors were of the opinion that the symptoms were caused by the constriction and not by adhesion to the intrathoracic structures (CLELAND; COLE, KEETON & WEBER; FRIEDBERG; HARRINGTON; HEUER & STEWART; HOLMES SELLORS; McMICHAEL; OPPENHEIMER, HITZIG & NEUHOF; SÖDERLUND; TUDOR EDWARDS & BARLOW; WESTERMANN; WHITE, ALEXANDER, CHURCHILL & SWEET). It was therefore considered necessary to decorticate the ventricular region. BLALOCK & BURWELL once again stressed that decortication should be started over the left ventricle, since it is easier to find a plane of dissection here. Moreover, there is less risk of pulmonary oedema when the left ventricle is de-

corticated before the right, as it is not necessary to fear that the pulmonary circulation will be overburdened with blood that could be pumped in from the freed right ventricle. Not only the pericardium but also the thickened epicardium should be removed. Presumably on account of the risk of injury to the atria, these authors, as well as HEUER & STEWART, stated that they need never be touched, nor was it necessary to free the venae cavae.

HOLMES SELLORS stated that decortication should never be carried beyond the region that can be surveyed or is easily accessible.

HOLMAN *et al.* (1949) held a somewhat different view on constrictive pericarditis to that of BURWELL and his successors. HOLMAN stressed that two factors are of importance for the results of surgical treatment. Firstly, there must be good access to all the areas requiring decortication, and this necessitates a sternum-splitting incision. Secondly, decortication of the heart must be adequate, with freeing especially of its right side, as well as of the inferior and superior vena cava. According to HOLMAN, the strongly pulsating left ventricle does not have such a great tendency to become encased as does the right side. The latter, with its thin vessels and atrial walls, more easily becomes compressed by the constrictive process. But he also stressed the importance of decorticating around the borders of the heart, since if decortication is incomplete, the heart is still enclosed in an iron grip, and the results are marred. HOLMAN's statements were based on experimental studies.

LAWRENCE, ADAMS & CASSELS (1948) reported a case in which constrictive pericarditis was localized to the pulmonary veins; it produced obstruction of these vessels on the left side, and led to pulmonary oedema and death. At autopsy, the pulmonary veins were found to be considerably stenosed where they passed through the pericardium, but the authors were of the opinion that a congenital malformation of these vessels might have been a contributory factor.

That the same process which involved the pericardium could also cause great impairment of respiratory function if it spread to the pleura was demonstrated by BURWELL & AYER (1941), who reported a case with bilateral pleural thickening. After transient improvement following operation for constrictive pericarditis, exacerbation and death ensued, owing to respiratory insufficiency. The authors drew a parallel between the pericardial effect on the heart, and that of the pleural lesion on the lungs. Both interfere with filling.

BECK had stated in 1937 that the constriction produced atrophy of

the heart; in 1941 he reported, together with ROBERTS, the results of post-mortem studies of the compressed heart. The diameter of the cardiac muscle fibres was found to be reduced by 30 per cent, and agreement with experimental studies was observed. The variability in size of the muscle fibres characteristic of the normal heart disappeared, so that all the fibres had the same, narrower diameter. These observations were in conformity with the results of HOSLER & WILLIAMS (1936), who found that the heart was of normal size or smaller than normal when extensive pericardial adhesions were present, but no valvular or vascular lesions could be observed. The largest hearts were, without exception, observed in the presence of rheumatic pericarditis. The authors expressed the view that the adhesions do not, in themselves, produce any circulatory disturbances, unless they cause compression, angulation or rotation of the heart.

The form of surgical treatment was established, in that all were in agreement that decortication should be performed. Only the extent of decortication and its site remained open to discussion. In practice, however — irrespective of the view held — the method adopted for decortication was that most likely to produce the best results. Consequently, HOLMAN's view that the site of entry of the caval veins and the right side of the heart should be decorticated, but that decortication should be continued beyond the cardiac borders, became in practice fairly similar to the functional results when the ventricles were decorticated. In both cases, the impediment to ventricular filling was removed, and this was presumably the essential factor for success of the operation.

When cardiac catheterization was introduced for clinical purposes, it was natural for interest to become focused on the haemodynamic conditions in constrictive pericarditis before and after operation. This was stressed by WHITE, ALEXANDER, CHURCHILL & SWEET, who were of the opinion that this method of examination had thrown new light on both the disease and the unsuccessful results obtained earlier. They found the pulmonary artery pressure to be raised in two of three operated cases, this rise confirming constriction of the left ventricle, which had the same effect on the pulmonary circulation and the right ventricle as mitral stenosis.

In 1946, BLOOMFIELD, LAUSON, COURNAND, BREED & RICHARDS published pressure curves from the right side of the heart in chronic constrictive pericarditis. They described the double-peaked curve in the

atrial tracing, and the diastolic dip in the ventricular curve, but did not make any mention of the pulmonary artery pressure in this disease.

As early as 1902, LORRAIN SMITH & MCKISACK had observed the increase in blood volume and total haemoglobin in a case of chronic constrictive pericarditis. This was subsequently confirmed by GIBSON & EVANS, BURWELL *et al.* and STEWART and his co-workers. The increase in blood volume was similar to that occurring in other forms of congestive heart disease.

Accumulation of blood in the peripheral veins was an early symptom to be observed; it was interpreted as damming-up of the blood in the veins owing to the impediment to their emptying into the atrium, due to chronic pericarditis. Little had been written about the conditions in the pulmonary circulation, since pressure measurements at this site could be performed only in animal experiments. It was occasionally mentioned that the pulmonary fields were clear, and that no pulmonary stasis was present. VOLHARD & SCHMIEDEN did not speak of pulmonary stasis until the left side of the heart had become incompetent, which occurs at a late stage. FERRARI (1930) stated that signs of pulmonary oedema were sometimes found at autopsy. It was not, in fact, until the 1950's that it was realized that the raised pressure in the pulmonary circulation, observed by WHITE and his co-workers, was due to the conditions being similar in both sides of the heart.

A conception of the pressure in the left ventricle was provided by measuring the pulmonary capillary venous pressure (PCV); these investigations were made by HELLEMS, HAYNES & DEXTER (1949), LAGERLÖF & WERKÖ (1949) and DOW & GORLING (1950). The rise in pressure in the pulmonary circulation could be ascribed to the left-sided constriction. This resulted in interest being transferred from the right ventricle to the left.

SAWYER (1950) and BURWELL (1951) measured the PCV pressure, the diastolic pressure in the pulmonary artery, the end-diastolic pressure in the right ventricle and the diastolic pressure in the atrium, and found an approximately similar rise in all of them. BURWELL established that the pressure in the pulmonary vessels is high in chronic pericarditis, and that there is pulmonary congestion of similar type to that in mitral stenosis or left-sided heart failure. The similarity between the pressure in the systemic veins and the PCV pressure indicated that the right ventricle no longer participates to the usual extent in ejecting the blood. Constriction of the opening of the great veins into the heart or the tricuspid ori-

fice had never been observed to result from a local fall in pressure at examination. The pulmonary congestion contributing to dyspnoea in pericardial disease, as well as the raised venous pressure, were an indication that the function of both ventricles was impaired by the pericardial changes. This was regarded to indicate that it was unnecessary to decorticate other parts of the heart than the ventricles.

The difference between pulmonary stasis in constrictive pericarditis and in mitral stenosis was stated by BURWELL to be that, in the latter, the right ventricle is able, owing to hypertrophy, to produce rapid, marked changes in the degree of pulmonary stasis, with resulting pulmonary symptoms. In constrictive pericarditis, the right ventricle is incapable of producing such changes, and this is why acute or paroxysmal symptoms so seldom appear. SCANNEL, MYERS & FRIEDLICH (1952) were able in five cases to confirm pulmonary hypertension by cardiac catheterization.

In 1952, SAWYER *et al.* reported their views on the circulatory consequences of the pericardial lesions. Their patients were examined both at rest and during slight exertion. The results with respect to stroke volume and cardiac output were in agreement with those of earlier workers. Pressure measurements in the pulmonary circulation in particular showed raised pressure, which increased still more on exertion. Postoperatively, some normalization of these pressures occurred, but restitution was not complete. The resistance in both the pulmonary and systemic circulation was found to be normal. The authors stated that the circulatory impediment was to be sought chiefly in the diastolic obstacle to ventricular filling, but common sense indicated that, if the pericardium was transformed into a hard casing or was adherent to its surroundings and to the heart, systolic activity could also be impaired. The pericardial changes were not, however, regarded to be solely responsible, since improvement but not full recovery was observed in many patients operated on. A persisting increase in the filling pressure after decortication was considered to indicate incomplete decortication of the ventricles, which could be corrected. Slow recovery indicated that a myocardial factor, hard to evaluate, was also present, with atrophy and infiltration of connective tissue as a result of spreading of the inflammatory process to the myocardium, as had been observed at autopsy.

The difficulty of dealing with the myocardial factor, in comparison to the mechanical effect exerted by the pericardium, was demonstrated by HARVEY *et al.* (1953). Of five patients, only one was operated on for

constrictive pericarditis; two patients had signs of myocardial involvement, which was a factor contributing to the picture of congestive heart failure. The authors evidently ascribed great importance to the favourable reaction to digitalis and conservative treatment as a criterion of the role played by the myocardium, and no operation was therefore performed in the remaining cases.

SUMMARY

It has been considered that the circulatory impediment in chronic constrictive pericarditis consists either of the blood finding difficulty in entering the right side of the heart, or of the inability of the ventricles to receive sufficient blood. Consequently, the attitude to surgical treatment of the disease has undergone a corresponding change. It is nevertheless possible to discern that most workers have abandoned the belief that any impediment to inflow of the blood exists. The salient feature underlying the haemodynamic disturbance is the inability of the ventricles to relax adequately in diastole and possibly, in advanced cases, to contract sufficiently in systole. Consequently, decortication is applied mainly to the ventricular region, only its extent remaining a debatable question.

EXPERIMENTAL STUDIES ON THE INFLUENCE OF CONSTRICTIVE PERICARDITIS ON CARDIAC FUNCTION

It had been known ever since the middle of the 16th century that absence of the pericardium does not necessarily imply any essential disturbances. Consequently, the prerequisites for pericardectomy were, in fact, established, particularly since it was inconceivable that the abnormal pericardium could perform any of the functions of the normal pericardium. These had been studied by BARNARD (1898), KUNO, EVANS & MATSUOKA (1915) and YAMADA (1917), and the functions under pathologic conditions by ROSE (1884) and by CONHEIM & STARLING (1889). However, since many of these experiments indicated that the pericardium had an important, vital function, further experiments were made to determine whether or not the heart could manage without it. AMERIO (1895) found that the resected margins of the pericardium became adherent to the surroundings. In total pericardectomy, no adhesions could be seen between the heart and adjacent organs. He did not find pericar-

dectomy to have any effect on the life of the experimental animals, and therefore concluded that the pericardium could be removed in human subjects. PARLAVECHIO (1909), MAZZONE (1912) and D'AGATHA (1912) removed the pericardium in dogs, and found that the functional disturbances observed were not incompatible with life.

Further experimental contributions to pericardectomy were made by REHN (1913) and by KLOSE (1921). They excised parts of the pericardium, and grafted autogenous fat or omentum into the defects, to re-establish continuity and prevent the pericardium from becoming adherent to the heart. REHN induced pericardial adhesions with aleuronat or tincture of iodine, and stated that the tension produced in the pericardium by fluid and adhesions impaired outflow from the inferior vena cava in particular, with resulting stasis in the portal region and the liver. Stasis in the vena cava was compensated by the development of collaterals, to the azygos vein among others. The pleural effusion observed in experiments on dogs was interpreted as an impediment to outflow from the pulmonary veins. REHN stated that injection of iodipin and oil of sesame into the pericardium was consistently able to prevent the severe adhesions produced by the irritant substances, after the initial exudative pericarditis.

The method used by FLESCH & SCHOSSBERGER (1906) and REHN (1913), *i. e.*, inducement of pericardial lesions by irritant substances, was later adopted by many workers who studied the changes associated with chronic constrictive pericarditis. Mention can be made of DIBLE (1921), BECK *et al.* (1925), HERRMAN & MUSSEY (1928), OCHSNER & HERRMAN (1929), FERRARI (1930), BLALOCK & BURWELL (1935), DIBLE & LYNCH (1938), YODICE (1941), FISHMAN *et al.* (1949), VOLWILER, BOLLMAN & GRINDLAY (1949), BOUCEK & GRINDLAY (1950), HOLMAN (1951), BOUCEK, GRINDLAY & BURCHELL (1952), DAVIES, LINDSAY & SOUTHWORTH (1952), EHRENHAFT & TABER (1952), ISAACS *et al.* (1952), and PARSONS & HOLMAN (1955). Different tissue-irritating substances were used, and their mode of application varied with the experimental conditions.

Three different types of investigation can be distinguished. In one of them, the object was to ascertain whether extrapericardial adhesions were able to produce any signs of heart failure. In the second type, a study was made of the influence of the pericardial and extrapericardial changes. Finally, the effect was studied of local constriction, and of the results of decortication.

HERRMAN & MUSSEY (1928) introduced various irritant substances

through a pericardiotomy, which produced marked anterior mediastinitis, and sometimes pericardial adhesions. This resulted in cardiac hypertrophy, which was considered to be due to the greatly increased burden on the heart. In the following year, HERRMAN repeated the experiment, together with OCHSNER, and obtained the same results. The pericardial adhesions were regarded to be the most important factor. The object of the investigation was to determine whether fresh adhesions would form after decortication according to Délorme, and endanger the results. They claimed that intrapericardial administration of a digestant, after the adhesions had been freed, prevented formation of new adhesions, with the exception of those due to reinfection at operation.

The effect of extrapericardial adhesions was also studied by BECK (1931) and HOSLER & WILLIAMS (1938). They sutured the pericardium to areas of varying size in the diaphragm, or to the anterior surface of the thoracic wall. No measurable circulatory changes were observed in any of the cases. BECK wounded the surface of the heart in various ways, in order to produce adhesions; except in the cases in which cardiac tamponade supervened, no measurable circulatory changes could be demonstrated. He therefore stated that, in all the experiments performed to produce extracardiac adhesions, he had seen no influence on the heart in the form of hypertrophy or dilatation, owing to the extra burden assumed to be placed on it by traction in the adhesions. If such a heart became hypertrophied, it was due to the valvular disease often associated with the adhesions. The only disturbances that could occur were due to angulation of the heart, or kinking of the great vessels at its base. Beck stated:

“Adhesions play no part in the production of compression syndromes. When present, adhesions are silent and incidental findings and produce no circulatory trouble whatsoever, unless the heart is acutely angulated or twisted. Our experimental and clinical evidence for these statements is conclusive and leaves no room for doubt.”

Irritant substances in the pericardium were found to produce distinct constrictive pericarditis, with liver lesions, ascites and oedema. FERRARI injected alcohol or iodine solution, and BLALOCK & BURWELL aleuronat. BECK used Dakin's solution for this purpose, and YODICE used hydrochloric acid, collodium, heat, saline solution, aleuronat and iodized talc, among other measures.

When Dakin's solution, in which the irritant substance consists of free chlorine, was injected by BECK into the pericardium, it produced a strong reaction, with erosion of the blood vessels and haemorrhage, the end-results being highly reminiscent of constrictive pericarditis. A rise in

venous pressure and a decrease in cardiac output were the first signs to be observed. Ascites and oedema followed, and roentgenologic examination showed a diminution in the size of the heart, and decreased pulsations. Changes appeared in the QRS complexes on the electrocardiogram. The pericardial cavity was sometimes obliterated, but no circulatory disturbances were observed unless the pericardium was thickened. If this occurred, and the pericardium surrounded the heart with scar tissue without becoming adherent to it, the typical syndrome appeared. It included a fibrinous covering on the liver, which had the appearance of the classical Zuckergussleber. BECK stated that there was impairment not only of diastolic filling, but also of the coronary circulation. When the disease had become manifest, decortication was performed. The circulatory reserve was so poor in many of the animals owing to the large accumulation of fluid in the abdominal and thoracic cavities, that they died during induction of anaesthesia. This could be prevented by aspirating the fluid from the abdomen and thorax prior to operation. BECK followed SCHMIEDEN's suggestion, and decorticated the left side first, but he went further and decorticated the atria and venae cavae as well. These operations taught him a number of practical details, which could subsequently be put to good use in cardiac surgery in man.

GIBBON, HOPKINSON & CHURCHILL (1932) studied the effect of constriction of the pulmonary artery on arterial and venous pressure. They found that an effect did not appear until 60 per cent of the cross-section area was constricted. When more than 85 per cent of this area was involved, constriction was invariably fatal. It can be inferred from this investigation, as well as from later ones, that constriction of the great vessels in chronic constrictive pericarditis is not of such great importance as was originally believed. Moreover, as far as I am aware, no such marked constriction of the pulmonary artery has been mentioned in connexion with this disease. The authors drew no essential conclusions regarding this matter from their investigation.

The effect of the effusion in the pleural, abdominal and thoracic cavities was studied by BLALOCK & BURWELL (1935) and DIBLE & LYNCH (1938). When BLALOCK *et al.* had induced pericarditis by administration of aleuronat, with a resulting rise in venous pressure, they determined the pressure in the cerebrospinal fluid and the thoracic duct, which was dilated. The pressure was found to be raised, and a pulsating flow of lymph was expelled from the puncture hole in the thoracic duct on expiration. The raised lymphatic pressure, which was also observed by YODICE (1941),

was not regarded by the authors to be associated with the fluid in the serous cavities.

DIBLE & LYNCH investigated whether local venous obstruction could be responsible for hydrothorax. Nothing abnormal was found in the pulmonary veins examined by them. After a study of the absorption conditions in the mediastinum, they pointed out — on the basis of their own and others' experiments — that disturbances in capillary permeability played a role in the development of hydrothorax.

Cardiac tamponade was found to have a similar effect on the circulation to that of chronic constrictive pericarditis. The common factors were the rise in venous pressure and diastolic filling pressure, as well as the decrease in pulse pressure and cardiac output. This was pointed out by BECK, and by ADCOCK, LYONS & BARNWELL (1940) and EVANS, WALTER & HELLEMS (1950), among others.

As a rule, no difficulty had been encountered in producing constrictive pericarditis experimentally. The strongly irritant and traumatizing substances used in early investigations were subsequently replaced by various plastic products. One of their advantages was that they could be applied for local constriction of the heart, as demonstrated by DUNCAN & ISAACS (1947) and FISHMAN *et al.* (1949). Furthermore, they were not involved by the inflammatory reaction, but could easily be removed from the space between the thickened, fibrous surfaces of the pericardium.

BOUCEK & GRINDLAY, as well as BOUCEK, GRINDLAY & BURCHELL, and VOLWILER, BOLLMAN & GRINDLAY, chiefly studied the effect of obstruction to the inflow on the venous and arterial side of the heart, respectively. The inflow to the right side of the heart was impeded by constriction of the venae cavae. The venous pressure was found to change inappreciably or not at all during the early period of circulatory failure, but to rise gradually as ascites increased. The arterial blood pressure was low, and glomerular filtration, renal plasma flow and diuresis showed an initial parallel reduction, which was rapidly normalized. Ascites and oedema developed, and a connective tissue covering appeared on the liver. Despite the high venous pressure, both blood volume and serum protein were normal. The ascitic fluid had a high protein content, which was assumed to be due to an increased flow of lymph from the liver.

In constriction of the inflow to the arterial side of the heart, pulmonary oedema was the salient feature. Circulatory failure was characterized by hyperpnoea, hypoxia and an increase in blood volume. The disturbances in renal function disappeared rapidly, whereas chloride excretion

was consistently low. There was apt to be salt retention, which accentuated the symptoms. The blood volume increased, and vascular changes appeared in the lungs, owing to increased pressure in the pulmonary circulation.

These two syndromes of right-sided and left-sided failure, respectively, appeared either separately, or — if pericarditis was produced by Dakin's solution, resulting in bilateral constriction — concurrently. Normal conditions were restored by decortication. Autopsy was performed on the dogs in which ascites had appeared. The thoracic duct was found to be considerably dilated, and the lymph capillaries of the liver enlarged. Since renal function was unimpaired, it was considered that it could have played no role in the development of ascites. This was explained by the high pressure in the systemic veins preventing the thoracic duct from emptying; the lymph therefore leaked out or burst the vessels, and collected in the form of ascites. The protein content of the ascitic fluid was high, particularly the albumin fraction, and similar values were found in the hepatic lymph. The authors regarded these findings to be in agreement with those of BLALOCK & BURWELL and of VOLWILER, BOLLMAN & GRINDLAY.

DAVIS, LINDSAY & SOUTHWORTH implanted cellophane in the pericardial cavity; oedema predominated during the acute stage, and ascites during the chronic stage. They studied the electrolyte and fluid balance and renal function during both stages. The early postoperative fluid and salt retention was ascribed to adrenal activity, whereas ascites was regarded to develop on a hydrostatic basis.

One of the foremost contributions to elucidation of the effects of constrictive pericarditis was made by ISAACS *et al.* in 1952. In this disease, it is not only the mechanical action of the pericardium on circulation that must be taken into account, but also fibrosis and atrophy of the myocardium, which constitute a factor that is hard to evaluate. It is difficult to determine which of these two factors is the more important.

One of the objects of the investigation made by ISAACS *et al.* was to produce a purely pericardial lesion, in order to ascertain its consequence when the myocardium was intact or inappreciably damaged. The other object was to determine the pathophysiologic implications of scars at different sites in the heart. Volume-elasticity curves were obtained post mortem by correlating the filling pressure with the diastolic blood volume. When the ventricle was full, greater pressure was required to produce a small volume increase, since the myocardium offered greater elastic

resistance. When the pericardium was thickened, still greater pressure than usually was required. The change in volume elasticity was the first indication that higher pressure was necessary to fill the ventricle with sufficient blood to maintain cardiac output. An index of the filling handicap was the rise in end-diastolic pressure in the ventricle, as well as the rise in atrial, venous and pulmonary capillary pressure. The authors considered it probable that this rise was due to changes in blood volume and volume elasticity in the vessels and blood reservoirs.

Constriction was produced by suturing the pericardium to the myocardium, and introducing the irritant substance into the pockets thus formed.

The following inferences can be drawn from the investigation of ISAACS *et al.* If the volume of the pericardium is diminished and its elasticity impaired, as in chronic constrictive pericarditis, it forms an impediment to normal diastolic filling of the ventricles. The high systemic venous pressure indicates the degree to which filling of the right ventricle is impeded, and the high pulmonary capillary pressure the corresponding impediment in the left ventricle. Consequently, when the whole heart is constricted, both these pressures are raised.

Constriction of the right atrium was found to produce no circulatory disturbances. As an estimate of the degree of atrial constriction required to have an effect on the circulation, the authors made a comparison with the investigations of GORLIN & HAYNES on the mitral orifice. The latter workers found that a decrease in its size amounting to almost 70 per cent was necessary to produce any symptoms on exertion.

ISAACS *et al.* considered it most likely theoretically that constriction of the right atrium would assume such proportions that it would be reflected in the circulation.

They found that when the right side of the heart was decorticated, the venous symptoms regressed, but pulmonary hypertension persisted, and the dogs died of pulmonary oedema. If the left side was decorticated, pulmonary stasis disappeared, but the symptoms of venous stasis remained, and the animals died without pulmonary oedema. Decortication of the right atrium alone had no effect. Decortication of the whole heart with the exception of the right atrium restored normal conditions, as did total decortication.

HOLMAN and PARSONS & HOLMAN induced pericarditis in a similar way to that of ISAACS *et al.*, and studied the effect of the local obstruction. HOLMAN focused main interest on the right side of the heart. In his experiments, he observed the changes typical of constriction of the inferior vena cava or of the right side of the heart, and regarded this as proof of the site of the circulatory disturbance. The inferior vena cava was more easily involved owing to the erect position; a reduction amounting to half its cross-section was, however, necessary to produce any symptoms. Constriction of the right atrium resulted in no circulatory disturbances. Acute constriction of the pulmonary veins was associated with pulmonary oedema and hydrothorax, and led to death; chronic constriction produced neither pulmonary oedema nor hydrothorax. Constriction of the left side of the heart was found to result in slight pleural effusion, decreased aortic pressure and diminution of cardiac output. Constriction of the left atrium produced no symptoms. The author therefore concluded that constriction of the right side of the heart was the most important factor. It was recommended that rings in the atrioventricular region be removed, and decortication include the venae cavae if this region was constricted. Late regression of ascites in operated cases was ascribed to formation of collaterals when decortication was inadequate.

SUMMARY

It can be inferred from the clinical and experimental evidence reviewed in this chapter that the main effect of constrictive pericarditis is to hamper ventricular function, particularly diastolic expansion. Local obstruction is seldom the cause of circulatory disturbances. Cardiac catheterization is the method of choice for determining both the presence of local constriction, and the extent of the constrictive process. This information is decisive for the extent of decortication. The role played by myocardial involvement is stressed, but it is pointed out that no definite or reliable means exists for evaluating the contribution of this factor to the constriction syndrome.

CHAPTER 2

AETIOLOGY

A. Earlier Investigations

1. AUTOPSY MATERIAL

The results of the underlying process, often acute pericarditis, can range from insignificant adhesions to thickening and contraction of the layers of the pericardium, interfering in one way or another with the heart action. Acute pericarditis is more often overlooked than diagnosed; WALLGREN (1945) found, in fact, that only one in every three cases was diagnosed ante mortem. SMALLEY & RUDDOCK (1946) and SAVILAHTI (1949) came to the same conclusion. They stated that the disease might appear as a relatively mild complication of some more serious illness, and was then not particularly characteristic. However, even in cases in which the basic disease was mild, and pericarditis was the dominating feature of the clinical picture, it could remain undetected. Consequently, it is understandable that the search for a plausible connexion between a manifest pathologic syndrome and its presumptive cause is associated with great uncertainty. Sometimes one disease and sometimes another has been favoured by different authors as being responsible for the constriction, but in many cases even a thorough investigation has failed to reveal the cause. It has sometimes been thought possible to trace a connexion between an injury to the chest and constrictive pericarditis. Although it has been a standing question whether rheumatic infection leads to constriction, no final answer has yet emerged from the discussion.

In constrictive pericarditis, the symptoms result from the effect of the mechanical factor constituted by the pericardium, and the functional factor constituted by the myocardium. Adherent pericardium does not,

in fact, necessarily imply constriction. It can be agreed with DETERLING & HUMPHREYS (1953) that a study of the literature reveals fairly great discrepancies in the views on what should actually be counted as constrictive pericarditis. The term constrictive implies an effect on the heart that is both anatomic and functional. It is, however, no easy matter to infer, from the large autopsy series in which mention is made of the relation of the pericardial adhesions to acute pericarditis, either the aetiology of patho-anatomically evident constriction or its symptoms. Interest has often been focused chiefly on the size of the heart in the various forms of pericardial adhesion.

Acute pericarditis, which is unanimously agreed to be the precursor of constriction, may appear at any age, but is more common in the lower age groups. Its incidence is higher in men (WESTERMANN 1944). The ratio of men to women is stated by SAVILAHTI to be 3:2, by SMITH & WILLIUS (1932) to be 2.3:1 and by WHITE to be 3:1. In autopsy series, its incidence varies between 4.2 per cent (SMITH & WILLIUS), 11.9 per cent (MUSSEY & HERRMAN 1926) and 16.6 per cent (LOCKE, cit. SMITH & WILLIUS). An average value is 9.2 per cent of 15,363 autopsies.

According to SMITH & WILLIUS, unclassified pericardial adhesions were present in 38.4 per cent of all cases of pericarditis, which comprised 4.2 per cent of 8,912 autopsies. Similar lesions were observed by LYTER (1920) in 5.8 per cent of 514 consecutive autopsies, by MOSCHKOWITZ (1953) in 1.4 per cent of 9,618, by SAVILAHTI in 1.9 per cent of 3,054, and by SPRAGUE, BIRCH & WHITE (1932) in 2.15 per cent of 1,953 autopsies. The incidence of pericardial adhesions is also higher in men than in women. LASSEN (1937) has stated the ratio to be 1.3:1, SAVILAHTI 5:2 and SPRAGUE, BIRCH & WHITE 1.7:1.

Thus, pericardial adhesions can be estimated to develop in roughly one-third of all cases of acute pericarditis. The fatal outcome in these cases is to be ascribed not so much to the pericardial changes, but rather to lesions of the myocardium and endocardium. If these cases are excluded, a small number nevertheless remains in which the patho-anatomic changes are fully compatible with the features of chronic constrictive pericarditis, although a connecting link between symptoms and findings at autopsy is most often lacking.

TUBERCULOSIS VERSUS RHEUMATISM

The morbid anatomic picture is characterized by thickening of the pericardial layers, and the presence of encapsulated, fluid-filled cavities, as well as calcifications. This has been largely responsible for the condition being interpreted as the final stage of tuberculosis, as described by OSLER in 1893, even when there has been no histologic or bacteriologic evidence in favour of such a diagnosis. BROOKS & LIPPENCOTT (1909), in their study of pericarditis, observed tuberculosis in 17 cases of the chronic adhesive form, but rheumatism in only 4 cases. In none of these cases was death caused by pericarditis, which was regarded to have played an inappreciable role in the prognosis. Tuberculosis of the pericardium was discussed more from the point of view of whether it was primary or secondary. WELLS (1901), who denied that the pericardium could be the primary and only localization, stated that tuberculosis could often lead to synechia and adherent pericardium, but that the circulatory disturbances were produced as often by rheumatism as by tuberculosis.

During the 10-year period 1921—1931, SPRAGUE, BIRCH & WHITE found, among 1,900 autopsies and 43 cases of pericardial adhesions due chiefly to rheumatism, no case that could be denoted as constrictive pericarditis. They did not consider tuberculosis to be responsible for the benign adhesions that were seen. During the 1897—1913 period they noted, in 3,053 autopsies and 62 cases of adherent pericardium, only three cases of constrictive pericarditis, all caused by tuberculosis. Of the 6 cases with a history of rheumatism, none had any rheumatic valvular disease, nor any signs of constriction. SMITH & WILLIUS were able to determine the aetiologic factor in only half of their cases of adhesive pericarditis, and found rheumatism and intrathoracic infections to be the most common causes. Only 39.5 per cent had had any cardiac symptoms or signs which, in four-fifths of these cases, were due to other lesions than pericarditis. In the remaining one-fifth in which constriction could be anticipated, the aetiologic factor was uncertain.

SAVILAHTI (1949) found only six cases of constriction in 59 cases of adherent or constricted pericardium. The constrictive forms were present chiefly in the groups with rheumatic heart disease, tuberculosis or polyserositis. The rheumatic adhesions were inappreciable, and the lesions could possibly be regarded as constrictive in two cases. Constriction had led to heart failure in those cases in which the cause was polyserositis or tuberculosis.

The fact that rheumatism is a common cause of pericardial adhesions was confirmed by MOSCHKOWITZ (1953); in 71 cases of adherent pericarditis, he found rheumatic fever to be the cause in 53.5 per cent, and tuberculosis in only 4.2 per cent. Constriction was present in only 7 cases, the cause being unknown in 3 of them. Two cases denoted as presumptive constriction were due to tuberculosis. One of the two remaining cases — both of polyserositis — was of rheumatic origin. The other had an unusual aetiology in the form of non-specific polyserositis with coincident endocarditis, which was regarded to be the remarkable feature.

It has long been known that the rheumatic infection is often a pancarditis, involving the myocardium, pericardium and endocardium. This naturally plays a great role from the prognostic point of view, compared to those cases in which the only essential disease is the mechanical constriction by the pericardium. Most authors have considered it unusual for the tuberculous infection to extend to the myocardium. HEIMANN & BINDER (1940), who were of the opinion that the black races are more liable to tuberculous pericarditis, found in their autopsy studies of Bantu negroes that the myocardium was fairly often involved as well, even when it was not a question of miliary tuberculosis. This had also been observed by OSLER, who stated that it was an exceedingly rare occurrence, and involved only the atria. The presence of tubercles in the myocardium or interstitial myocarditis was reported by WELLS, by BELLET, McMILLAN & GOULEY (1934), by SUZMAN (1943) and by PEEL (1948). Consequently, it is not possible in tuberculous pericarditis to rule out myocardial involvement.

SUMMARY

It can be inferred from the studies of autopsy material that rheumatic fever in the form of rheumatic polyserositis may play some role in the development of constrictive pericarditis, although it cannot as a rule be regarded as an important aetiological factor. Tuberculosis and intrathoracic infections are responsible for a large number of the constrictions, whereas in many cases the cause is unknown. Myocardial lesions may appear as a result of both tuberculous and rheumatic infection.

2. CLINICAL MATERIAL

If the studies on autopsy material do not provide much information of value regarding the aetiology of constrictive pericarditis, a survey of the clinical series is more illuminating.

RHEUMATISM

A study of the causes of constrictive pericarditis shows that many authors have — perhaps unjustifiably — denied that rheumatic fever can be responsible for the disease. As far as the symptoms of constriction on a rheumatic basis are concerned, a distinction must be made between two factors. One is the actual effect of constriction, and the other is the result of the rheumatic myocarditis and endocarditis. BECK (1931), who was among those to study the effect of the diseased pericardium on the size of the heart, found that constrictive pericarditis in the true meaning of the term was not invariably present, since restitution of function did not occur after decortication. Consequently, the size of the heart was not related to the rheumatic constriction, but was more dependent on the rheumatic valvular disease. Improvement following pericardectomy was to be attributed to hypertrophy of the heart (ALLEN & GRAHAM 1929, GIBBON & CHURCHILL 1931). Heart failure was stated to be due to the valvular disease, the adhesions ascribed to the rheumatic infection being too inappreciable to give rise to circulatory impairment.

Another factor contributing to this view was the follow-up studies made in cases of rheumatic pericarditis. HOLT (1929) investigated the final effect of the rheumatic infection in a series of children with a definite history of rheumatic pericarditis. She found pericarditis to be only one feature of a progressive heart disease, with a valvular defect, usually mitral stenosis and sometimes aortic incompetence, so that none of the children recovered completely. Constriction was not mentioned as a cause of the symptoms and signs of incompetence, which were regarded to be fully explained by the heart disease. The same indications were provided by the investigations of WHITE (1935) and of BLAND (cit. HARRISON & WHITE 1942) who, in a follow-up study of 1,500 cases of rheumatic heart disease, observed not a single case of constrictive pericarditis. JAKOBSSON (1946) also investigated the end-results of rheumatic pericarditis. In no case did he find clinical, electrocardiographic or roentgenologic evidence of constriction.

SCHMIEDEN & FISCHER (1926) stressed the importance of rheumatism in the causation of constrictive pericarditis, and attributed a prominent role to rheumatic fever. Rheumatism, angina and infectious diseases were, in fact, found to be the aetiologic agents in 68 per cent of their operated cases, investigated by WESTERMANN (1944). LIEBIG (1929), who followed up 8 of 11 children who underwent cardiolysis according to BRAUER's method, noted rheumatism in 5 cases. Constrictive pericarditis was, however, lacking in one case, as could be confirmed at autopsy, and was not particularly conspicuous in the other cases. PÄSSLER (1933) made a follow-up investigation of 19 patients operated on by REHN & SCHMIEDEN, and found the origin to be rheumatic in 10 of them.

ROTHSTEIN (1934) collected 34 cases from the literature, in order to compare the effect of cardiolysis with that of decortication; 17 out of 27 cases were considered to be produced by rheumatism. TUDOR EDWARDS & BARLOW (1946) noted a possibly rheumatic origin in 5 of 20 cases. They did not, however, regard a history of rheumatic fever as proof that it was responsible for the subsequent pericarditis. The presence of pus and granulation tissue was, in their opinion, more indicative of tuberculosis.

CHAMBLISS *et al.* (1951) assembled 61 cases of constrictive pericarditis, with a history of rheumatic fever in 10 of them. In only one of these cases was it possible that constriction had resulted from the rheumatic process. KALTMAN *et al.* (1953) reported 18 cases of constrictive pericarditis; in 5 cases with enlargement of the heart and signs of valvular disease, rheumatism might have been the only causative factor. An additional three patients had a history of recurrent rheumatic fever, but no signs of rheumatic heart disease.

MOSCHKOWITZ reported that in one of 7 cases the rheumatic process led to polyserositis, which produced constriction. DETERLING & HUMPHREYS had only one case of conceivably rheumatic origin in 25 definite cases of constrictive pericarditis. In a series of cases of pericarditis diagnosed clinically, or established at operation or autopsy, only one of the 139 cases of rheumatic origin exhibited a clinical picture and post-mortem features consistent with chronic constrictive pericarditis. KROOK (1954) reported 16 cases of constrictive pericarditis, the diagnosis being confirmed at operation in 11 of them; in one case the symptoms had their onset following a recurrence of rheumatic fever (see Table I).

Table I. Incidence of some of the common causes of constrictive pericarditis in the literature.

Author	Rheumatism	Tuberculosis	Unknown
ANDREWS <i>et al.</i>		16/18	
BLALOCK & BURWELL		18/20 (28)	
BÖRGER		7/9	
CHAMBLISS <i>et al.</i>	1/61	16/61	38/61
DACOSTA		9/10	
DETERLING & HUMPHREYS	1/25	8/25	12/25
EDWARDS & BARLOW	5/20	11/20	
HARRINGTON		5/24	19/24
HARRISON & WHITE		5/28 (37)	29/37
WHITE 1935		10/15	
HARTL		8/8	
HEUER & STEWART		1/18	
HOLMAN & WILLETT		4/9	5/9
KALTMAN <i>et al.</i>	5—8/18		7/18
KROOK	1/16	3/16	
McKUSICK		8/20	
LIEBIG	5/11		
MOSCHKOWITZ	1/7		
VAN NIEUWENHUIZEN		5/35	
OPPENHEIM <i>et al.</i>			5/7
PAPO		34/38	
PÄSSLER	10/19		6/19
PAUL <i>et al.</i>		9/42 (53)	21/42
ROTHSTEIN		9/27	
STEPHANS		2/4	
WESTERMANN	36/54	18/54	10/54
Total	68/231	206/501	152/296

TUBERCULOSIS

A number of authors are unanimous in stating that rheumatism is not a cause of constrictive pericarditis, but that tuberculosis is of more importance as an aetiologic factor (ANDREWS, PICKERING & SELLORS; ARMSTRONG; BECK, BLALOCK & LEVY; BURWELL & BLALOCK; CHAMBLISS *et al.*; HARRINGTON; HARRISON & WHITE; HEUER & STEWART; HOLMAN & WILLET; MORTENSON & WARBURG; TUDOR EDWARDS & BARLOW). It is not, however, always such an easy matter to confirm the diagnosis, even when a presumptive causal relation exists. A history of tuberculosis, and signs of its presence at other sites in the body, can presumably be accepted as probable evidence that pericarditis is of tuberculous origin. A negative report from the pathologist on specimens taken at operation does not rule out such an origin, in view of the fact that an earlier active infection may heal without traces (PAUL, CASTLEMAN & WHITE). Moreover, it is known that tuberculosis of the pericardium may heal without residual symptoms, since it has been observed

at autopsy on patients who died of some other disease (HEIMANN & BINDER 1940).

BLALOCK & LEVY found, in 42 patients under observation for tuberculous pericarditis at the Vanderbilt Hospital, that pericardectomy had been performed in 6 of the 24 certain cases, and might have been of value in an additional two. Decortication was indicated in one of the 18 suspected but unconfirmed cases. It was considered that constriction would supervene in 8 of every 24 cases of tuberculous pericarditis. A higher figure for this occurrence was given by ANDREWS, PICKERING & SELLORS, *i. e.*, 16 out of 18 cases, and by STEPHANS (1951), who stated that 2 of 4 cases of tuberculous pericarditis end in constriction. CARROL (1951) investigated HARVEY & WHITEHILL'S 71 cases of tuberculous pericarditis, and found that 14 were alive five years after the onset. He added another 7 patients, all of whom had been under observation for an average 12 1/2 years after the onset of tuberculous pericarditis. Electrocardiographic changes appeared in three and calcification of the pericardium in one, but operation for constrictive pericarditis was necessary in only one case. If no distinction is made between confirmed and presumptive cases, WESTERMANN found tuberculosis to be the causative factor in 34 per cent of VOLHARD & SCHMIEDEN'S operated cases of constrictive pericarditis (*cf.* Table I).

A rough survey of the literature shows that tuberculosis was responsible for 206 of 501 cases of chronic constrictive pericarditis. This is a higher incidence than that reported by CHAMBLISS *et al.*, who found tuberculosis to be the aetiologic factor in 28 per cent of their series. It is not so long ago that no effective therapeutic media were available for tuberculosis; consequently, this disease had an important prognostic role in the earlier cases (CHAMBLISS *et al.*). BLALOCK & LEVY had a mortality rate of 83 per cent in their cases of definite tuberculosis of the pericardium. HARVEY & WHITEHALL had the same experience, and CHURCHILL warned against operation during the acute stage, since the results were disappointing. BECK made the following statement: "As long as the tuberculosis shows activity, one cannot expect to relieve the cardiac compression". Although the situation has been altered by the advent of antibiotic and chemotherapeutic agents, only the future can show what the final results will be.

INFECTION

Suppurative pericarditis may lead to constrictive pericarditis, but does so only sporadically. BLALOCK (1940) stated that this is the end-result of pyogenic infections that are not drained. Staphylococci or pneumococci are the most common infecting organisms (BLALOCK & BURWELL; HARRISON & WHITE; KOLETSKY & BROWNLEE; LILIENTHAL; SHIPLEY & WINSLOW; SODEMAN, among others). It is of some interest to note that constrictive pericarditis did not develop in the cases of streptococcal pericarditis in DETERLING & HUMPHREY's series, described by MOORE (1935), despite the fact that the pericardium was irrigated with Dakin's solution. For, BECK has stated that the use of Dakin's solution for this purpose leads to constrictive pericarditis.

A non-suppurative form of pericarditis may appear in connexion with an upper respiratory tract infection, and lead to constrictive pericarditis in some cases. It has been regarded as a virus infection, or as suspected tuberculosis, but it is difficult to obtain any definite proof of the aetiology in such cases. Operated cases in which the only finding was an illness of this nature have been mentioned by HARRINGTON, by PAUL *et al.* and by SELLORS.

Other infectious diseases have sometimes been known to give rise to pericarditis, which may occasionally result in constriction. This has been regarded to apply to syphilis, but it is difficult to find any definite report of constriction on a syphilitic basis. *Bacterium tularensense* and *Actinomyces bovis* are other possible aetiological agents, which must be mentioned for the sake of completeness. JAGER & RANSMEIER (1943) had one case caused by tularaemia, and traced another nine cases in the literature. HARA & PIERCE (1957) described cases of pericardial constriction in which *Actinomyces bovis* was the infecting organism.

An inflammatory reaction in the pericardium may produce pericarditis, and when the reaction has been sufficiently marked, it has been known to lead to constriction. This can be inferred from the animal experiments made with a view to studying constrictive pericarditis. It is therefore somewhat surprising that none of the surgeons who applied asbestos or talc to the pericardium, in order to produce vascular adhesions for the treatment of angina pectoris, observed constrictive pericarditis during a follow-up period of 14 years (BECK & THOMPSON, cit. DETERLING & HUMPHREYS).

TUMOURS

A tumour of the pericardium as a link in a metastasizing neoplasm of the thoracic wall or the lungs is not an especially uncommon phenomenon. The metastatic process is seldom so extensive that constriction appears, but death has been reported from acute cardiac tamponade, as a result of the rapidly increasing pericardial exudate. WALLACE & LOGUE (1946) described a case in which constrictive pericarditis developed in bronchial carcinoma with metastases, and SLATTER, KROOP & ZUCKERMAN (1952) a case in which it appeared 14 years after operation for carcinoma of the breast. In the latter case, roentgen therapy had produced fibrosis of the mediastinum, which may have been a contributory factor.

METABOLIC PROCESSES

Metabolic processes may be a cause of pericarditis, the most common example being the uraemic pericarditis which may appear terminally in nephritis. Compression can occur, but not constriction. Cholesterol pericarditis is a rare disease, which is generally seen in association with myxoedema. Among those to report the presence of large quantities of cholesterol in the pericardial exudate are ALEXANDER (1919), GORDON (1929), ADA *et al.* (1950) and CREECH *et al.* (1955). CREECH *et al.* described a case in which constrictive pericarditis developed, and pericardectomy was performed. The authors ascribed the constriction to the high cholesterol content of the pericardial fluid. The cause of this accumulation of cholesterol is uncertain.

TRAUMA

It is known that a penetrating or non-penetrating injury to the chest may be the cause of cardiac disorders. This may occur either by extravasation of blood into the pericardial cavity (BECK; BERBER; BERNSTEIN), or by involvement of both the myocardium and endocardium (WARBURG). The acute haemorrhage in the pericardium, together with the injury, produces the signs of compression described by BECK in one of his two compression triads, but it is not established beyond question whether haemopericardium alone can be the cause of constriction. Experiments in dogs have not provided a definite answer. REHN (1913), BECK & MOORE (1925) and EHRENHAFT & TABER (1952) injected autologous blood into the pericardial cavity in dogs, but in none of their ex-

periments did they observe any serious changes, nor were signs of constriction ever seen. EHRENHAFT & TABER also injected the lipid fraction of the blood into the pericardium, and this resulted in somewhat greater changes. The longer the observation period, the greater were the changes. Symptoms of stasis appeared in one of the dogs as a sign of constriction, and it died after seven weeks.

Even if animal experiments have been unable to elucidate the relation between haemopericardium and constriction, a sufficient number of cases of non-penetrating injuries has accumulated to establish an incontrovertible causal connexion. In 1940, GLENN (cit. DETERLING & HUMPHREYS) reported the development of constriction three years after injury by the steering wheel of an automobile. At autopsy, no other cause could be detected. Many other authors have observed cases in which constrictive pericarditis followed a non-penetrating injury to the chest (BARKER & JOHN-SON; BLALOCK; DEJOU; DETERLING & HUMPHREYS; MORTENSON & WARBURG; OVERHOLT *et al.*; WHITE & GLENDY).

In penetrating injuries in the heart region, greater interest is focused — as DETERLING & HUMPHREYS have pointed out — on immediate treatment and survival of the patient than on follow-up examinations. In order to ascertain whether constrictive pericarditis supervened in any cases of this nature, the aforementioned authors questioned those surgeons who could be assumed to have the greatest experience of such matters. It was found that, in many cases, the answers could not provide the information desired. In such injuries, it was difficult to determine whether the essential cause of constriction was the presence of blood in the pericardial cavity, or the coincident contamination by bacteria and the possibility of bacterial pericarditis. A good illustration is given by the case described by RAVITCH. A penetrating wound of the pericardium resulted in pericardial effusion, for which repeated aspiration was performed. The pericarditis later proved to be tuberculous, and the knife causing the wound was considered to have passed through a tuberculous region, since active pulmonary tuberculosis was present.

STRAUSS reported the development of constrictive pericarditis, verified at operation, six years after cardiac resuscitation; it was caused by a broken-off injection needle which had remained in the heart and pericardium. Cases of constriction following gunshot wounds were observed by TUTTLE (cit. DETERLING & HUMPHREYS) and by MORTENSON & WARBURG. HARKEN, who had removed many foreign bodies from the heart region, had seen no instance of subsequent constrictive pericarditis, nor

had BECK, BIGGER or BLALOCK observed its occurrence after penetrating wounds of the heart (DETERLING & HUMPHREYS).

Penetrating injuries to the pericardium can be caused by swallowed foreign bodies in the oesophagus, which penetrate both this structure and the pericardium. However, here as well, the coincident infection is perhaps the most important factor in the causation of pericarditis. I have seen a case in which a foreign body (a needle) was extracted from the pericardium, where a large haemorrhagic exudate was present. This observation undoubtedly causes one to wonder to what extent undiagnosed foreign bodies in the oesophagus can have similar consequences.

Injection of sclerosants into oesophageal varices may lead to perforation of the pericardium, followed by pericarditis of a suppurative nature. I have seen a case in which injection led to development of a fistula between oesophagus and pericardium, which resulted in suppurative pericarditis with a fatal outcome.

POLYSEROSITIS

Polyserositis is an exceedingly indefinite condition, both as a disease and from the aetiologic point of view. MOSCHKOWITZ described a case which was found at autopsy to be constrictive pericarditis on the basis of polyserositis with atypical endocarditis. Since pericardium, pleura and peritoneum were involved in cases of tuberculosis and rheumatism, polyserositis proved to be the cause of completely adherent pericardium in more than half of his autopsy cases. The causes of adherent and constricted pericardium are closely related, and MOSCHKOWITZ stressed the importance of polyserositis as a causative factor in constrictive pericarditis. WOOD & KRUMBHAAR (1951) denied that polyserositis could be the cause of constrictive pericarditis, whereas ANDREWS *et al.* and HARRISON & WHITE maintained the contrary.

UNKNOWN FACTORS

Despite attempts to trace a putative cause, a large group of cases cannot be assigned to any aetiologic category, owing to insufficient evidence. Many authors have taken this as an indication that tuberculosis is implicated. In some cases it has been possible to trace an antecedent pericarditis following an infection of the upper respiratory tract, and this has been denoted as idiopathic. In other cases constriction has been the initial symptom. The series of various authors contain a relatively large pro-

portion of such cases, all equally obscure from the aetiologic point of view (see Table I). It is possible that acute idiopathic pericarditis, which is so apt to be overlooked, may have been the forerunner in cases in which constriction was primary. However this may be, it throws no additional light on this category of cases, and it must be accepted that their cause cannot be established.

During the period in which cases of constrictive pericarditis have been observed and treated, the introduction of chemotherapeutics and antibiotics for the treatment of infections has constituted a great advance. This must imply that cases with an earlier poor prognosis can now survive the acute stage. Since acute pericarditis can be regarded as responsible for constrictive pericarditis in many cases, some change can be assumed to have taken place in the incidence of the various aetiologic factors. It remains to be seen whether chemotherapeutic and antibiotic agents, owing to their bacteriostatic action, will decrease the number of cases of constriction, or whether they will increase them, by saving cases that were earlier fatal. The investigation of GRIFFITH & WALLACE (1949) indicates that other factors may also be implicated. They found that, in the course of 50 years, the number of cases of tuberculous and pneumococcal pericarditis had decreased. A high incidence of cases on a rheumatic basis was noted, as well as an increase in those of idiopathic origin. A definite increase was recorded in the incidence of cases due to uraemia, coronary disease and malignant processes.

B. Present Investigation

In the present series of 38 operated cases of constrictive pericarditis, distinct lesions typical of this disease were present in all the cases. Histologic examination of the resected pericardium was made in every case.

TUBERCULOSIS

Tuberculosis, either suspected or confirmed, must be assumed to be the cause in 15 cases. A history of tuberculosis and signs of this disease at other sites in the body were regarded as definite proof that constrictive pericarditis was of tuberculous origin. All these patients showed a positive reaction to the tuberculin test.

Four patients (Cases 4, 9, 14 and 31) had contracted erythema nodosum,

followed by polyserositis. Another four (Cases 19, 21, 28 and 34) had a history of pericarditis on a tuberculous basis. One patient (Case 20) had suffered from polyserositis of uncertain origin. At decortication, an intrapericardial abscess containing creamy pus was found; it was regarded as tuberculous, and guinea-pig tests on the postoperative exudate confirmed the diagnosis. As in MOORE & MURPHY's case, streptomycin therapy had a good effect. One patient (Case 26) had marked tuberculous manifestations, in the form of pulmonary tuberculosis and lupus, and tuberculosis had necessitated bilateral epididymectomy.

One patient (Case 23), denoted as probably tuberculous, had suffered from erythema nodosum 20 years earlier. The present illness had its onset in the form of acute pericarditis, with a strongly positive tuberculin reaction and raised antistreptolysin titre. Her general condition was remarkably unaffected during the acute stage. Death after operation was due to heart failure; at autopsy, pulmonary tuberculosis was found to be the only possible cause of constrictive pericarditis. In the other case of probably tuberculous aetiology (Case 25) there was a marked familial predisposition to tuberculosis. The patient had a history of polyserositis, presumably of tuberculous origin; it had produced not only constrictive pericarditis, but also constricting pleural lesions, with great impairment of pulmonary function. One patient (Case 35), in whom constriction was the initial symptom, had a strong hereditary tendency to tuberculosis, and a number of probably tuberculous pulmonary illnesses in his history. Postoperatively, pulmonary tuberculosis developed in the earlier inactive areas; it has later been treated successfully with anti-infective agents.

The last two (Cases 30 and 37) are fairly similar from the aetiological point of view. Lobectomy had been performed in both cases on account of pulmonary tuberculosis; acute tuberculous pericarditis had supervened, and on healing had produced the symptoms of constriction. In one of them, in whom the right lower lobe had been resected, bronchial tuberculosis gave rise to a fistula, following an abscess around the bronchial stump. The fistula had healed before pericardectomy was performed. In the other case, the right upper lobe had been resected; a fistula containing tubercle bacilli persisted in the operation scar. Both pre- and postoperative treatment were complicated in this case by a marked allergic diathesis to antibiotics, for example, especially to those intended for treatment of the tuberculosis. Even when the allergy to antibiotics did not prevent medication, the patient had a pronounced antipathy to taking those drugs that could be thought to have an effect on the tuberculous infec-

tion. Pulmonary tuberculosis was bilateral but stationary. The patient's condition nevertheless deteriorated gradually after decortication, and although an improvement in circulation was noted, he died of progressive pulmonary tuberculosis.

The latter case illustrates the poor prognosis, mentioned earlier by BECK and others, unless adequate anti-tuberculous therapy can be given. In both these cases, the relation between tuberculous pericarditis, pulmonary tuberculosis and operation is apparent.

In performing resection on tuberculous lungs, the possibility of infecting the surroundings with organisms from the tuberculous tissue is evident. The procedure is relatively risk-free if antibiotics can be given, and the most surprising fact is that complications so seldom occur. A particularly great risk exists if the pericardium is injured in one way or another. Part of the pulmonary vessels are covered by the pericardium, and more or less distinct extensions can be observed over the pulmonary veins in particular. If the pericardium is opened inadvertently, there is a risk that the bacteria which must always be assumed to be present in the operative field will enter it, and gradually cause infection at this site. Consequently, when operating on tuberculous lungs, the utmost care should be taken to avoid injury to the pericardium. Should this occur, it is of prime importance to deal with the lesion, after local application of antibiotics to the pericardium. Despite these precautions, the risk of pericarditis developing is great, and prophylaxis is the best form of treatment. I have seen three cases in which opening of the pericardium in the course of lung resection for tuberculosis produced, during the postoperative course, distinct electrocardiographic and clinical signs of acute pericarditis. In two of the cases, the condition yielded to treatment, and no signs of constriction have yet been observed. The third patient died of suppurative pericarditis.

Microscopic examination of tissues removed at operation confirmed the presence of tuberculous lesions of the pericardium in four cases (Cases 14, 19, 28 and 37). In the remaining 11 cases, the specimens consisted of chronic, hyalinized connective tissue, without signs of active infection; calcifications were visible macroscopically or microscopically in 9 cases. Preoperatively, the tuberculous aetiology could be confirmed by demonstration of tubercle bacilli in 3 cases. During the postoperative course, tubercle bacilli were recovered from the exudate in one case, and their presence verified by tests on guinea-pigs.

RHEUMATISM

A rheumatic origin was plausible in 4 patients (Cases 8, 10, 33 and 38). In two cases the onset was in the form of rheumatic fever with rheumatic polyserositis, and in two in the form of joint involvement. None of these patients had auscultatory signs of rheumatic valvular disease. Two of the patients in this group died after operation, one of them after temporary improvement lasting for a month. Autopsy verified the poor state of the myocardium, and the presence of an envelope of calcified tissue on the right side. The other death in this group is presumably to be ascribed to anoxaemic damage, owing to underventilation in connexion with operation and after it.

INFECTION

The disease appeared in connexion with infectious diseases of various origin and degrees of severity in 7 cases (Cases 2, 3, 12, 15, 16, 17 and 24). The relation between the infectious condition and possible coincident pericarditis was not always evident. Among the infectious diseases were measles, pneumonia, paratyphoid fever and typhoid fever.

Two cases in this group (Cases 2 and 15) exhibited a slight superficial clinical similarity. Both patients had a markedly myxoedematous appearance. The basal metabolic rate was low; the serum cholesterol content was known in only one of the cases, in which it was normal. In one case thyroid medication produced considerable reduction of the oedema; in the other it had no effect. It is not clear whether, in the former case, pericarditis had any aetiologic factor in common with the cholesterol pericarditis mentioned by ADA *et al.* In neither case did microscopic examination show any specific changes.

One patient (Case 24) had a history of two life-threatening illnesses resembling paratyphoid fever, and another (Case 12) had contracted typhoid fever in a German concentration camp.

UNKNOWN FACTORS

No aetiologic factor could be established in 8 patients (Cases 6, 7, 11, 18, 22, 27, 29 and 32). Five of these patients had acute pericarditis, and one of them had had distinct signs of life-threatening cardiac tamponade, which had not been diagnosed. No connexion with a previous infection existed in these cases. Naturally, an infectious factor cannot invariably

be ruled out in such cases, either in the form of tuberculosis or of a less serious infection. The tuberculin reaction was known in 5 of these 8 patients (Cases 6, 7, 18, 22 and 29); it was negative in only one of them (Case 18). In the remaining 3 (Cases 11, 27 and 32) no information on this matter was available. Distinct lesions of the lung parenchyma, which might have been an indication that pericarditis was of tuberculous origin, were present in only one patient (Case 29). Pulmonary stasis was, however, so marked that it could not be excluded that the changes observed were associated with it. With regression of pulmonary stasis, the greater part of the parenchymal changes interpreted as tuberculous disappeared. Guinea-pig tests on both sputum and pleural exudate were negative. A rheumatic origin was suspected in one patient (Case 7), owing to a heart murmur.

IDIOPATHIC

Acute pericarditis in connexion with infections of the upper respiratory tract was present in three cases (1, 13 and 36). All three were tuberculin-positive. Acute pericarditis with an upper respiratory tract infection as the only symptom has been classified as idiopathic, and tentatively ascribed to a virus infection. Unclear cases of different origin are undoubtedly present in this group, and the positive tuberculin reaction suggests the possibility of tuberculosis as one of the causes. In one of these patients (Case 13), calcification of the pericardium developed three months after the acute pericarditis.

TRAUMA

A traumatic origin was conceivable in only one patient in the whole series (Case 5). Eight years before the symptoms of pericarditis appeared, he received an injury to the left side of the chest, with fracture of the ribs. No other known cause of constriction was found in the history.

ACUTE PERICARDITIS.—A history of acute pericarditis was present in 24 cases. In 13 cases it was tuberculous. In the other groups, five can be noted in those of unknown aetiology, three in the idiopathic group, *i. e.*, all cases, two in those of rheumatic origin, and one in the group of infectious origin.

TUBERCULIN REACTION.—The tuberculin reaction was tested in 33 patients. In the remaining 5, it could not be ascertained whether or not the test had been made. Only one of those tested was tuberculin-negative;

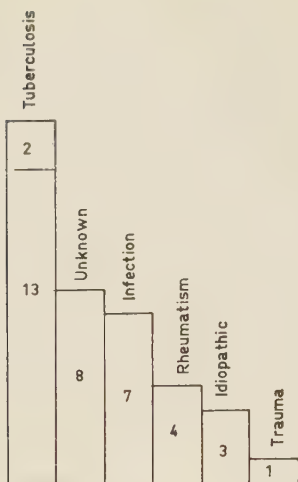


Fig. 1. Aetiologic factors in the present series: 38 cases of constrictive pericarditis.

he belonged to the group of infectious origin. Thus, a possibly tuberculous aetiology existed in at least 32 of the 38 cases of chronic constrictive pericarditis, whereas such an origin was less likely in one case. In 23 cases, the fluid from the operated side was examined for the presence of tubercle bacilli, with positive results in guinea-pigs in only one (Case 20).

HISTOLOGIC EXAMINATION.—The pericardium resected at operation was examined microscopically in every case. In conformity with general experience, this examination was not, as a rule, helpful in establishing the aetiology. In only four cases were tuberculous lesions observed. In the other cases, the specimen consisted of hyalinized, collagenous connective tissue containing numerous fibrils, with perivascular round-cell infiltrations and no signs of specific granulation tissue. In most cases, calcifications of varying size were also present in the tissue.

SUMMARY

In a series of 38 operated cases of constrictive pericarditis, tuberculosis is found to be the most common aetiological factor, it being responsible for almost three-eighths of the cases. Although a number of processes are regarded to be able to produce chronic constrictive pericarditis, some

cases are found in which evidence of any specific cause is lacking. In the present series, 8 cases belong to this category. In 7 cases there was an antecedent infectious disease. Three patients have a history of acute idiopathic pericarditis, and a rheumatic infection is a possible cause in 4. In only one case is there a conceivable connexion between constrictive pericarditis and an injury to the chest (Fig. 1).

CHAPTER 3

METHODS

LABORATORY TESTS

The patients were examined on an empty stomach, and in most cases the analyses were made at the clinical laboratory of Sabbatsbergs Sjukhus. In those cases in which the normal values and their range of variation are not shown in the relevant tables, they did not differ from those accepted as normal (Kliniska laborationsmetoder, Astra, Södertälje 1955).

SPIROMETRY

The spirometric studies were made at Sabbatsbergs Sjukhus. The technique described by BIRATH (1944) was used for determination of the residual capacity. Since absolute values are unsatisfactory as a basis of comparison, the ratio of observed values to normal values was calculated. The normal values were computed with the formulas given by BALDWIN, COUNNAND & RICHARDS (1948).

The ratio of residual volume to total lung capacity increases with age. It is about 20 per cent in healthy young males, and somewhat higher in females. BALDWIN, COUNNAND & RICHARDS (1949) and MOTLEY (1952) stated that a ratio of residual volume to total lung capacity exceeding 35 per cent must be regarded as a manifestation of pulmonary emphysema "in clinically significant degree". A higher ratio may, however, be normal in an elderly subject, since the 35 per cent limit must not be considered absolute in such subjects, who may have readings as high as 50 per cent without signs of pulmonary insufficiency or disability (COMROE *et al.* 1953).

At the laboratory where the spirometric investigations were carried out, the accuracy of the method has been discussed by UGGLA (1957).

CIRCULATION TIME

The circulation time was determined by rapid injection into an antecubital vein of 5 ml of a 20 per cent solution of decholin. The time was recorded between the injection and the instant the patient felt a bitter taste. The duration of the bitter taste was not recorded consistently. The normal range of circulation time from the antecubital vein to the tongue is 8 to 16 seconds.

CARDIAC CATHETERIZATION

The patient was examined on an empty stomach, and usually without the administration of a sedative. The pulmonary artery was catheterized, and an arterial puncture needle introduced into the brachial artery of the other arm.

The catheter was advanced from the pulmonary artery into the PCV position, and the pressure recorded. It was then withdrawn into the pulmonary artery, and the cardiac output determined at rest and after 7 minutes' work. Work was continued for altogether 10 to 13 minutes, when the pressure was recorded again. After 15 minutes at rest, or until the pulmonary artery pressure had regained the pre-working level, the catheter was withdrawn into the superior vena cava during continuous recording of the pressure.

Although this procedure sometimes had to be modified, owing to difficulties in performing catheterization, it was followed as closely as possible, in order to obtain pressure measurements under comparable conditions.

The catheter was connected to a drip infusion containing 1 g of procaine and 50 mg of heparin per 1,000 ml of physiologic saline solution. During the examination, an inappreciable quantity of this solution was flushed through the catheter, to keep it open when the pressure was not being recorded.

The work, which in most cases did not exceed 150 kgm/min, was performed on a bicycle ergometer. This was connected to the examination table, so that the patient — in the recumbent position — could tramp on the pedals in a fixed rhythm given by a metronome. Different work loads were obtained by varying the degree of braking on the wheel.

After catheterization, the catheter was withdrawn in some cases into the superior vena cava, slightly above the level of the right atrium, and

angiocardiography performed directly. In other cases, catheterization had to be repeated for this examination to be carried out.

COMPLICATIONS IN PREOPERATIVE CATHETERIZATION. — The complications which occurred were due to the intravasal manipulations. Any superficial thrombophlebitis was of a mild nature and required no special treatment. Deep thrombosis of an arm vein developed in one patient (Case 15); heparin therapy was given and it cleared up without any sequelae.

Complications in the form of knotting of the catheter were far more serious and troublesome. Judging by the literature, such complications seem to be uncommon, but it is likely that they have often failed to be reported.

It is sometimes difficult to advance the catheter in the desired direction, and repeated rotations in one or other direction are necessary. In constrictive pericarditis with calcifications, it may be impossible to visualize the catheter in the dilated right ventricle. Manipulations aimed at advancing the catheter into the pulmonary artery are therefore apt to lead to formation of loops in the ventricle, which may become actual knots if they are not detected, since movements of the catheter help to tighten them.

Such a complication occurred in Case 17. The catheter could be withdrawn from the heart under fluoroscopy and thence into the superior vena cava, without any discomfort to the patient beyond moderate pain on passage of the knot through the arm vein. Catheterization could be completed through the same vein without mishap. After withdrawal, the knot in the catheter had the appearance of an overhand figure of eight.

In Case 32, cardiac catheterization was followed by the appearance of pneumonia-like changes in the left lower lobe and tubercle bacilli in the sputum. Tuberculosis was the cause of constrictive pericarditis in this case. Operation could be performed after a course of appropriate therapy.

A brief spell of unconsciousness during catheterization occurred in two cases. Recovery was rapid, with no deleterious effects in either case. In one of the cases (Case 13) unconsciousness occurred after injection of decholin into the pulmonary artery, and was accompanied by generalized convulsions and cyanosis. Oxygen administration produced rapid recovery, and catheterization could be completed without further complications. The other patient (Case 30) became unconscious when the catheter passed into the pulmonary artery; it was presumably due to a disturbance in heart rhythm, so that the low cardiac output decreased still more and

produced transient cerebral anoxia. The ECG recording was forgotten during the time the patient was affected. In this case as well, recovery was rapid and catheterization could be completed.

Cardiac arrhythmias, usually ventricular extrasystoles, are not uncommon on passage of the catheter through the right ventricle. These disturbances are transient, and cease when the catheter has reached the pulmonary artery.

COMPLICATIONS IN POSTOPERATIVE CATHETERIZATION.—These occurred in three cases. In one (Case 16) definite signs of thrombophlebitis appeared.

In two cases knotting of the catheter occurred, but it could be removed in both cases without any serious damage to the patient. In Case 17, the same situation arose as at preoperative examination; the catheter became tied in a knot, and could not be removed without general anaesthesia, since it was arrested at the level of the axilla. It was withdrawn without any great difficulty, and the examination could be completed. In this case, there was an additional complication, in the form of an arterial thrombosis of transient nature in the arm. A short-acting barbiturate was injected into the arm not used for catheterization. An arterial puncture needle was already inserted in this arm, and the barbiturate was presumably injected through this needle. A few days after the examination, the patient had a tender cord along the course of the radial artery, and no pulsations could be felt in this artery at the wrist. Irritation by the barbiturate had thus produced thrombosis of the vessel. It gave rise to no symptoms, apart from the initial tenderness. At follow-up examination, the circulatory conditions were normal in this arm, with strong pulsations in both the radial and ulnar artery.

In Case 9, catheterization was exceedingly difficult to perform, and at preoperative examination an experienced examiner was forced to refrain from other pressure measurements than those in the right atrium, despite lengthy and conscientious efforts to advance the catheter. Since it was desirable to know the pressure in the other parts of the heart after operation, the whole battery of aids was enlisted to advance the catheter into the required position. It was probably twisted too many times, so that it suddenly became knotted in the atrium. After withdrawing the knot into the superior vena cava it could not be untied and it was necessary, after the permissible time for fluoroscopy had elapsed, to remove the catheter under general anaesthesia. In this process, the last part of the basilic vein was stripped by the catheter.

No haematoma or signs of mediastinal haemorrhage appeared in either of these exceptional withdrawals of the catheter.

COMMENTS.—It is naturally open to discussion whether catheterization should be insisted on when the conditions for performing it are so unfavourable. As far as constrictive pericarditis is concerned, particularly when calcifications are present, the conditions for fluoroscopy are exceedingly disadvantageous. If any results are to be achieved, it is necessary to proceed with more than usual determination, since it is highly tempting to abandon the examination when the difficulties pile up.

STEADY STATE

The chief problem in recording cardiac haemodynamics is to ensure that the investigation reflects the condition known as the steady state. The criteria of the steady state have been discussed by UGGLA (1957), who made his investigations at the laboratory of Sabbatsbergs Sjukhus. According to DONALD *et al.* (1954, 1955), this state is reached after one minute in healthy subjects, and usually after two to three minutes in patients with heart disease.

In the present investigation, a satisfactory steady state was reached after the seventh minute of work. The final pressure readings were made after 10 to 13 minutes' work. The work load was usually 75 to 150 kgm/min. As a rule, the load was lower at the preoperative examination than at the postoperative.

In order to evaluate the basal conditions of the investigation, the observed oxygen uptake was compared to the predicted value (Fig. 9). It can be inferred from the figure that many patients were strictly basal at the preoperative examination. It proved more difficult to attain this state at the postoperative examination (Fig. 26).

PRESSURE MEASUREMENTS

In the early cases, an ordinary water manometer was used for recording both the peripheral venous pressure and the mean pressure in the different chambers of the heart (Cases 1—11, 13—15 and 38). The peripheral venous pressure was determined with the patient horizontal and the site of puncture, most simply an antecubital vein, at the level of the anterior axillary line. For the sake of comparison with the right atrial pressure, the water pressure has been converted into mm Hg.

In the later cases, use was made of a Tybjerg-Hansen condenser manometer, or a strain-gauge manometer of the Statham type, modified by Swema-Elema. The impulses from the manometers were recorded, via an amplifier, with an Elmqvist six-channel electrocardiograph (Clinic, Elema).

A plane 5 cm dorsal to Ludwig's angle was taken as the zero level for the measurements with all three types of manometer. Correction was made for the hydrostatic pressure when the manometer was not exactly level with the point of reference.

Before and after each pressure measurement, the Hansen manometer was checked several times against atmospheric pressure and calibrating pressures.

The procedure was the same with the strain-gauge manometer, except that the calibrating pressure was built into the amplifier by a resistor, equivalent to a known pressure, with a step control for selection of a suitable range.

One to three channels of the electrocardiograph were used for recording the ECG, and one for the phonocardiogram.

The mean pressure was calculated with the aid of a planimeter, or by electric filtration. The systolic and diastolic mean pressures were calculated for the same phase of the curve.

The pulse rate was determined from the ECG tracings.

INTRACARDIAC PRESSURE MEASUREMENTS AT REST

On the basis of numerous investigations, the following pressures are stated to be normal (BLOOMFIELD *et al.* 1946, BORDEN *et al.* 1948, HICKAM & CARGILL 1948, LAGERLÖF & WERKÖ 1948, RILEY *et al.* 1948, DEXTER *et al.* 1950, STORSTEIN 1951, ELIASCH *et al.* 1953, FOWLER *et al.* 1953, GEELEN 1953, DONALD *et al.* 1955, SEMB *et al.* 1955).

Pressure mm Hg

RA	RV		PA			PCV
	Syst.	Diast.	Syst.	Diast	Mean	
6	17—31.5	0.5—8	14—29	4—13	8—19	2—13

Good agreement is generally found between the data of various workers, the greatest differences usually being due to differences in the zero level.

PULMONARY ARTERY PRESSURE AT WORK

HICKAM & CARGILL (1948) investigated their patients during mild exercise, and found only an inappreciable rise in pulmonary artery pressure. RILEY *et al.*, who used a heavier load of work, also found no constant or significant rise in pulmonary artery pressure.

This is in contrast to the findings of DEXTER *et al.* (1951), who recorded a slight rise in pressure during mild exercise and a larger rise on greater exertion. DONALD *et al.* (1955) observed a greater rise in pressure, but they used a heavier load than earlier workers. In eight healthy subjects who performed work that can be characterized as light, the oxygen uptake not exceeding 400 ml/min/m², the pulmonary artery pressure rose from 18 to 24.6 mm Hg, which implies a rise of almost 37 per cent. SÖDERHOLM (1957) expressed the opinion that the mean pressure in the pulmonary artery at work should be less than 25 mm Hg with an oxygen uptake up to 500 ml/min/m².

The variations in pressure in the pulmonary circulation have been discussed by ELIASCH (1952) and UGGLA (1957), at the laboratories where the present investigations were carried out. UGGLA found a standard deviation in the individual case of 1.22 mm Hg at rest, and 1.68 mm Hg at work. ELIASCH regarded changes in the PCV pressure exceeding ± 4.7 mm Hg and changes in the mean pulmonary artery pressure exceeding ± 17.4 per cent as significant.

OXYGEN UPTAKE

The expired air was analyzed according to HALDANE. The errors in estimation of oxygen uptake have been analyzed by DONALD *et al.* (1955). HARVEY *et al.* (1949), DAHLSTRÖM (1950) and ELIASCH (1952) found a mean difference between duplicate determinations of oxygen uptake not exceeding 5 per cent. No duplicate determinations were made in the present series.

OXYGEN CONTENT OF BLOOD

The oxygen content of arterial and mixed venous blood was determined with the method of VAN SLYKE & O'NEILL (1924). At least two determinations were made on each sample. The analytic error of the arteriovenous oxygen difference was calculated as the difference between

the first and second determinations, and resulted in a standard deviation of 0.14 ml/L or 0.11 per cent for A-V oxygen differences ranging from 30 to 143 ml/L. This is in conformity with the results of UGGLA (1957), who used the laboratory of Sabbatsbergs Sjukhus, and those at the laboratory of St. Eriks Sjukhus, where part of the patients in the present series were investigated. No duplicate determinations of the A-V oxygen difference at rest and at work were made, nor was any correction made for physically dissolved oxygen. The error in determination of the oxygen capacity was added to the calculation of oxygen saturation.

The A-V oxygen difference in healthy subjects has been given by Cournand *et al.* (1945) as 45 ml/L. In the present series, an A-V oxygen difference exceeding 50 ml/L at rest has been denoted as pathologic, as stated by ELIASCH (1952).

HICKAM & CARGILL (1948) regarded an A-V oxygen difference on effort exceeding 69 ml/L at an oxygen uptake of less than 400 ml/min/m² as pathologic. Similar figures were reported by RILEY *et al.* (1948), DEXTER *et al.* (1950) and ELIASCH (1952).

According to Cournand *et al.*, the normal value for arterial oxygen saturation lies between 92 and 98 per cent. ELIASCH reported a mean value of 93.3 per cent and no saturation below 89 per cent without cardiopulmonary involvement, and BJÖRK *et al.* (1953) at the laboratory of Sabbatsbergs Sjukhus 94.0 per cent with a range of 89 to 98 per cent. The lower limit of normal range should, for older persons at any rate, be fixed at 92 per cent (BALDWIN, Cournand & RICHARDS 1948).

According to BJÖRK *et al.*, no change in arterial oxygen saturation or only slight elevation is produced by moderate work.

CARDIAC OUTPUT

The cardiac output (CO) was determined by the Fick method, and calculated from the formula:

$$\begin{aligned}\text{CO L/min} &= \frac{\text{O}_2 \text{ uptake ml/min}}{\text{A-V O}_2 \text{ difference ml/L}} \\ \text{Stroke volume} &= \frac{\text{CO ml/min}}{\text{Pulse rate beats/min}} \\ \text{Stroke index} &= \frac{\text{Stroke volume}}{\text{m}^2 \text{ body surface area}}\end{aligned}$$

No duplicate determinations of the cardiac output were available. The errors in determining the cardiac output with Fick's method are assumed not to exceed 15 per cent (HAMILTON *et al.* 1948, among others). The mean value for the normal stroke index was taken as 56 ± 1.9 ml. This figure was computed from a series of 23 normal subjects investigated by DONALD *et al.* (1955) and HOLMGREN *et al.* (1957); the lowest cardiac index in this series was 41 ml.

HEART VOLUME

The heart volume was measured roentgenologically with the patient standing, according to LILJESTRAND *et al.* (1939). Owing to the changes in blood distribution, the variations in heart volume are greater in the erect than in the recumbent position. This fact is, however, certainly of minor importance in the present series, since in chronic constrictive pericarditis the heart obviously has no room for dilatation within the limited space of the pericardium.

The following figures were used as the upper normal limits of the heart volume: males, 500 ml/m² of body surface area; females, 450 ml/m² and for the children in this series, 350 ml/m² of body surface area.

ANGIOCARDIOGRAPHY

In all except the earliest cases, angiocardiology was performed with synchronous depiction in the frontal and lateral projections on Elema's roll-film serial changer, constructed according to Gidlund's design. The contrast medium was usually injected with an automatic syringe on Gidlund's principle, through a catheter of Cournand or Lehman type (no. 9—11), introduced into the superior or inferior vena cava, close to its entry into the right atrium. The automatic syringe is linked to the film changer and starts it at the required time. The contrast medium used was a 70 per cent solution of Umbradil (Astra) or of Urokon in a dose of 1.1 ml per kg of body weight. The injection time ranged from 2 to 3.5 sec.

The exposure time was 0.03 to 0.06 sec at 350 mA and 85 to 125 kV. The rate of exposure was 2/sec in each plane for 15 to 25 sec, or 6/sec for the first 5 sec and 2/sec thereafter. The position of the exposures in the cardiac cycle was marked on the electrocardiogram recorded simultaneously. For further details of the technique, reference is made to KJELLBERG *et al.* (1955).

STATISTICAL METHODS

The statistical analyses were made with the methods currently used in medical statistics (FISHER 1954).

The significance of means and differences was tested with the t test, using the following levels of significance:

5 per cent $< p$:	not significant
1 per cent $< p < 5$ per cent:	almost significant
0.1 per cent $< p < 1$ per cent:	significant
$p < 0.1$ per cent:	highly significant

CHAPTER 4

CLINICAL FEATURES

1. GENERAL

One operation was performed on 37 of the 38 patients in the present series, and two operations on the remaining patient.

AGE AND SEX

The series consisted of 30 males and 8 females. The average age at operation was 38 years 5 months, ranging from 9 years 5 months to 63 years 7 months (see Fig. 2).

SYMPTOMS

INITIAL SYMPTOMS.—Dyspnoea, ascites, oedema or a combination of these symptoms, as well as easy fatiguability in a few cases, were the initial manifestations. In some cases the condition had been diagnosed earlier as cirrhosis of the liver, mitral disease or myocarditis.

The average duration of the symptoms was 7 1/2 years, the longest duration being 26 years and the shortest 3 months (see Fig. 3).

Since dyspnoea, ascites and oedema are the most common features of chronic cardiac compression, an analysis was made to determine the incidence of each of them as the initial symptom. Dyspnoea on exertion was found to have been the initial symptom in 21 cases, discomfort in the epigastrium and abdominal swelling in 7 cases, oedema in 5 cases, and dyspnoea at rest in 2 cases. One patient had a stabbing sensation in the precordium as the initial symptom, one fainting on exertion, and one had a feeling that the blood mounted to his head and caused suffocation.

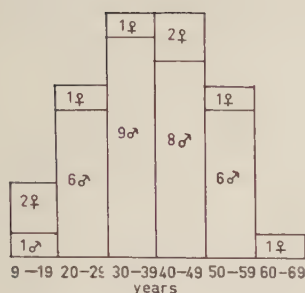


Fig. 2. Age and sex distribution in the present series (♂ = males, ♀ = females).



Fig. 3. Duration of the symptoms in the present series: 38 cases of constrictive pericarditis.

GENERAL SYMPTOMS.—The most common symptom was dyspnoea on exertion; it was present in 26 cases, and varied in severity from fairly inappreciable discomfort to complete disability. The average duration of dyspnoea was 4 years 8 months, ranging from 7 months to 26 years.

Oedema was present in 23 patients. It was initially periodic, and usually consisted of swelling of the ankles and feet in the daytime; later, it became permanent and of varying extent. One patient was unable to lie down, owing to pharyngeal oedema, with swelling of the uvula to twice its ordinary size in the morning. Three patients had oedema chiefly of the face, peripheral oedema being completely lacking. The average duration of oedema was 3 years 5 months, with a range of 3 months to 12 years.

Ascites was the next most common symptom. It was observed in 15 cases, and varied from slight to massive. The average duration was 4 years, the range of variation being 3 months to 14 1/2 years.

The least common symptom was dyspnoea at rest, which was present in 13 patients. Its average duration was 10 months, with a range of 2 months to 3 years.

Dyspnoea on exertion and oedema were thus the most common symptoms and, together with abdominal swelling, those of the longest duration. Dyspnoea at rest, present in 13 patients, was usually transient, and sometimes appeared periodically. Permanent dyspnoea at rest was observed in only five patients, as an indication of a copious pleural exudate or marked pulmonary stasis.

Increased fatiguability was common, and was disabling in some cases. Other prominent symptoms were tachycardia, precordial or substernal oppression, a stabbing sensation in the heart region, an unproductive cough, and dyspeptic complaints.

PARACENTESIS

Thirteen patients had undergone some form of paracentesis before operation, varying in the respective cases from 1 to 18 times. Altogether 91 aspirations had been performed; 54 of them consisted of laparocentesis, with removal of more than a total 130 litres of ascitic fluid, 23 of thoracocentesis, with removal of more than 44 litres, and 14 of pericardiocentesis, with aspiration of more than 4 litres of fluid. Laparocentesis had been performed 14 times in one patient in the course of 3 1/2 years, and another had undergone pericardiocentesis 10 times during a period of 4 1/2 months. Three cases required laparocentesis only, and two thoracocentesis only. All three cavities had to be aspirated in one patient in the course of six months, whereas two patients underwent both laparocentesis and pericardiocentesis, and three both laparocentesis and thoracocentesis.

2. PHYSICAL EXAMINATION

It has been stated that constrictive pericarditis should be strongly suspected if a young subject has signs of heart failure, with ascites and pleural effusion or peripheral oedema, as well as quiet or muffled heart sounds, only slight mobility of the cardiac borders at fluoroscopy, low blood pressure and pulse pressure, and raised venous pressure. A thorough examination should be made, with this diagnosis in mind. As VOLHARD pointed out as early as 1907, it is mainly the circulatory disturbances that are conspicuous, whereas the local cardiac findings are less prominent. He wrote: "... das auffallende Missverhältnis zwischen den hochgradigen offensichtlich kardialen Stauungserscheinungen und dem geringfügigen objektiven Herzbefund". VOLHARD introduced the term "Einflußstauung" for this central stasis which, in typical cases, is characterized by symptoms and signs that can be established by physical examination, *i. e.*, peripheral cyanosis, stasis in the neck veins, dyspnoea, ascites, and oedema of the legs.

GENERAL CONDITION

Since the illness had a duration of several years in many of the patients, it is not surprising that they were marked by it. Of the 38 patients, 17 were denoted at the first examination as being chronically ill. In one (Case 19), the clinical picture was characterized by an acute febrile state, with a dry cough, associated with a pericardial friction rub and crepitations at the base of both lungs. Successive exacerbation took place, but the condition gradually became stabilized, so that it did not differ markedly from what was denoted as chronic illness in the other cases.

VENOUS STASIS

Venous stasis is a common sign, and VOLHARD pointed out the distension of the neck veins, which do not empty in the upright position, often with a double collapse in systole and diastole. In the present series, venous stasis was present in 28 cases; it appeared as distinct distension of the veins of the neck, which did not collapse when the patient was upright. Measurements of the venous pressure were made in a few of the earliest cases, but this was subsequently replaced by recording of the atrial pressure. Examination for pulsations of the neck veins, interpreted as their collapse, was not made consistently, but in a few cases a note is found of pulsating neck veins. In 9 of the earliest cases, the venous pressure ranged from 14 to 27 mm Hg, with a mean value of 20.3 ± 1.5 mm Hg.

CYANOSIS

According to LUNDSGAARD & VAN SLYKE (1923), cyanosis appears in the presence of unsaturated haemoglobin in a concentration of at least 5 g/100 ml of blood. One of the causes of cyanosis is prolongation of the peripheral circulation time, which permits a greater degree of oxygen dissociation than usually. Cyanosis also appears when oxygen diffusion in the lungs is impaired, either by a reduction in the respiratory surface, or by pulmonary lesions, due to pulmonary hypertension. In the present series, cyanosis could be ascribed to all these factors, which were active to a varying extent in the individual case. Cyanosis was present in 20 patients, 9 of whom had a prolonged circulation time. In 10 cases the circulation time was not determined. In two cases, both low arterial oxygen saturation and prolongation of the circulation time were present.

CHAMBLISS *et al.* found that half of the patients who died during hospitalization were cyanotic before operation. In the present series, all but one of the patients who died after operation were cyanotic preoperatively.

DYSPNOEA

In view of the fact that dyspnoea is such a common feature of this disease, it is surprising that so few of the present patients were denoted as dyspnoeic at clinical examination. It can only be concluded that mild degrees of functional dyspnoea escaped the attention of the examiner. Consequently, a notation of dyspnoea at rest indicates a recorded observation, which can perhaps be regarded to have some significance. Five (Cases 8, 14, 16, 27 and 32) were dyspnoeic at rest. In two of them (Cases 8 and 14) dyspnoea was due to pleural lesions, with effusion in both pleurae, and in one (Case 32) to greatly reduced vital capacity, as a result of old pleural lesions and pulmonary hypertension. One of the two remaining patients (Case 27) had reduced respiratory function, owing to asthmatic pulmonary lesions, with low vital capacity and emphysema. The other (Case 16) in this group presumably had vascular pulmonary lesions, owing to pulmonary hypertension.

Thus, dyspnoea was not such a common symptom as could be expected, taking into account the often great reduction in pulmonary function, owing to the bilateral pleural exudate, or pulmonary lesions associated with pulmonary hypertension.

OEDEMA

Oedema was present in 16 cases. It was generally dependent, but sometimes appeared round the eyes, or in the throat, hampering respiration at night. The degree of severity varied greatly, and at the first examination in hospital, extensive oedema was observed in only three cases. Since one of the objects of preoperative treatment was to relieve the oedema, the patients' weight was recorded. During the preoperative period, 11 patients lost an average 4.6 kg in weight, ranging from 1.3 to 14.6 kg. Five patients gained an average 4.2 kg, the range being 0.9 to 7.3 kg. Obviously, other factors than excretion or accumulation of oedema fluid may have been responsible for the change in weight in these cases. Slight variations in weight during the preoperative period were noted in 17 patients, but no appreciable difference was found in comparison to

the weight on admission. In the remaining cases, no information was available on this matter.

In the patients with oedema, the mean value of the serum protein was 5.75 ± 0.41 per cent, with a value below 4 per cent in four cases. When no oedema was present, the mean serum protein was 6.88 ± 0.18 per cent. The difference is significant (0.1 per cent $< p < 1$ per cent).

In those cases in which the serum albumin and serum globulin were determined, no significant difference was present between the cases with and without oedema.

CHAMBLISS *et al.* found patients with oedema to have a 1 per cent lower value for the serum protein than patients without oedema. This observation is in agreement with the present investigation.

ABDOMINAL EXAMINATION

ENLARGEMENT OF THE LIVER.—The liver was enlarged in 36 patients. The size varied from a liver palpable about one fingerbreadth below the costal margin to one extending below the umbilical plane. The liver was sometimes hard to identify on palpation, owing to distension of the abdomen by varying quantities of ascitic fluid. After aspiration of the fluid, definite signs of liver enlargement were invariably observed in previously uncertain cases.

ASCITES.—Ascites was recorded in 15 patients, and was probably present in an additional two.

PALPABILITY OF THE SPLEEN.—The spleen was palpable in two cases.

PULSE RATE

Only one of the patients had fever at the first examination; he was admitted to hospital with acute pericarditis. All the others were afebrile on examination. In 12 cases the pulse rate at rest was 90/min or above. The highest rate at rest was 130/min, and the lowest 60/min, the mean value being 84 ± 2.4 /min.

A paradoxical pulse has long been regarded as a typical feature of constrictive pericarditis. It consists of a decrease in systolic and diastolic pressure during inspiration, with an increase during expiration. Thus, there is no irregularity in rhythm, but only in amplitude. The paradoxical pulse has been described by GAISBÖCK, by KATZ & GAUCHAT,

by KUSSMAUL, by VAN DER MANDELE, by ROSENBAACH, by SEMERAU, by SOMERBRODT, by WENCKEBACK and by WILLIAMS, among others. The true paradoxical pulse, which appears in all arteries, may be due to two factors. (1) An abnormal state in the respiratory system, in which the increased negative pressure in the pleura presumably plays the greatest role. (2) A pathologic condition of the pericardium, in which the normal intrapleural pressure is believed to affect the flow to the heart (KATZ & GAUCHAT). Normally, the heart, lungs and intrathoracic veins are affected simultaneously by the respiratory variations. But if the pericardium is thickened, the variations in pressure do not affect the filling pressure in the heart, and the paradoxical pulse arises on account of the slowing down of the blood in the lungs and the intrathoracic veins.

Examination for the occurrence of a paradoxical pulse was made in few of the present cases, and in none of them was there any note of such an observation. A paradoxical pulse was, however, recorded at cardiac catheterization in one patient (Case 18).

PULSE PRESSURE

Low pulse pressure, in addition to a high pulse rate at rest, has been regarded as characteristic of constrictive pericarditis. If 40 mm Hg is taken as a normal pulse pressure, 21 of the present patients had low pulse pressure. The values ranged from 15 to 35 mm Hg, with a mean value of 29 ± 1.26 mm Hg. In 17 cases the pulse pressure was 40 mm Hg or above, with a maximum of 70 mm, and a mean value of 48 ± 2.06 mm Hg.

AUSCULTATORY FINDINGS

BECK's description of constrictive pericarditis mentioned the small, quiet heart as an important characteristic. In the present series, a typical murmur was rare; it was present in only four cases, and was systolic in all of them. In one (Case 7) the murmur, together with the other clinical signs, was interpreted as due to mitral stenosis, which was probably incorrect. Mitral incompetence may have been responsible for the murmur, but unfortunately the function of the mitral orifice was not studied in connexion with operation.

The extra heart sound characteristic of constrictive pericarditis is a protodiastolic sound; it is most clearly audible over the centre of the heart, towards the apex, over the ventricular region. POTAIN (1856) was the first to report this extra sound. It was also described by LIAN, MARCHAL

& PAUTRAT (1933) and denoted as protodiastolic pericardial vibrations, which were stated to occur in the presence of pericardial calcifications. This view was strengthened by the pericardial calcifications found in their two cases. McKUSICK (1952) held the same view. EVANS (1943) mentioned the protodiastolic sound, but wished to replace the term gallop rhythm by protodiastolic triple heart rhythm. This appears as an added sound in early diastole, directly after the second sound. EVANS ascribed some importance to it as a diagnostic sign.

WOOD *et al.* (1951) stated that a diastolic heart beat occurred concurrently with the protodiastolic sound; it had been described earlier by BRAUER and by FRIEDREICH.

FROMENT *et al.* (1949) were able, in a case of calcified pericardium, to observe four heart sounds, one of which was audible in early diastole, and presumably corresponded to the aforementioned sound. However, they also heard an additional sound in early systole, and ascribed it to vibrations in the calcified surface of the pericardium, or the junction of articulated plaques.

The protodiastolic triple rhythm occurs as a third sound after the second heart sound, with an interval of up to 0.12 sec. It is heard most distinctly over a region between the left mamillary line and the left sternal margin, but is often audible over the greater part of the heart. By means of simultaneous pressure recordings and electrokymograms, it has been shown to occur concurrently with the rapid filling of the ventricle. It is found both in atrial fibrillation and with a regular heart rhythm. The position of this sound in the cardiac cycle is in good agreement with the explanation that it is due to vibrations in the muscle wall of the ventricle, caused by the impact of the inrushing blood. This protodiastolic triple rhythm was present in 17 of the present 38 cases.

THORACIC WALL

Great importance as a diagnostic sign has often been accorded to the thoracic-wall phenomena, which were among the first signs of constrictive pericarditis to be observed. Retraction of the thoracic wall in systole has been interpreted as a sign of the increased burden of work placed on the heart by its adhesion to intrathoracic structures. The heart is bound to the thoracic wall by the pericardial adhesions, and becomes immobilized between the spinal column behind it and the sternum in front of it. These

changes have been observed on the electrocardiogram, as well as at roentgenologic and physical examination.

HEIM described systolic retraction in the epigastric angle, and SKODA systolic retraction of the interspaces at the site of the apical impulse, whereas TRAUBE pointed out that such retraction could also be observed in the absence of pericardial adhesions. Systolic retraction not only of the interspaces, but of the whole anterior thoracic wall, including the ribs and sternum, was observed by FRIEDREICH; he ascribed it to adhesion of the heart to the diaphragm, its origin being retracted by the action of the heart. BROADBENT described systolic retraction between the eleventh and twelfth ribs to the left of the spinal column.

Additional phenomena in the heart region have been reported. Thus, MORGAGNI mentioned absence of the apical impulse, and WILLIAMS the distinct awareness by the patient of the heart action. Diastolic shock or "Schleudern" of the thoracic wall was reported by BRAUER and by FRIEDREICH. WENCKEBACK stated that the thoracic wall does not expand in the normal way during respiration. However, since in many cases these signs in the thoracic wall are by no means characteristic of the disease, their diagnostic value has come to be questioned. As early as 1923, VOLHARD & SCHMIEDEN warned against overestimating the importance of these features, which are so often lacking.

In the present series, systolic retraction was present in 8 cases. It was localized to the precordium, and in only one case is there mention of the parts of the thoracic wall involved. In this case, there was retraction of the whole thoracic wall over the precordium, including both the interspaces and the ribs. In the other cases, the size of the area in which systolic retraction was observed varied from the palm of a hand to smaller areas. Presumably, it was the interspaces that were retracted.

To sum up, it must be stressed that no single symptom or sign is decisive, but that the diagnosis is based on the clinical picture as a whole. Formerly, the phenomena observed in the thoracic wall contributed to distinguishing between the two forms of constrictive pericarditis, *i. e.*, accretio cordis and concretio cordis, for which different forms of treatment were recommended. However, as VOLHARD pointed out, it is rare to find either form isolated, but the variants are only differences in the degree of constricted pericardium, and lack any essential importance. When the symptoms call for treatment, it is the same in both forms, namely, decortication.

CHAPTER 5

LABORATORY STUDIES

1. BLOOD

SEDIMENTATION RATE

The erythrocyte sedimentation rate (S. R.) was determined in all but one case. The mean value was 18 ± 4 mm/1 hr, ranging from 1 to 80 mm/1 hr. The S. R. was over 16 mm/1 hr in 11 cases. In two of them, the rise was due to active pericardial tuberculosis, whereas in the others there was no demonstrable cause, apart from the change in serum protein associated with the circulatory disturbance.

RED CELL COUNT AND HAEMOGLOBIN

The values for the red cell count (RBC) and haemoglobin (Hb) in all 38 cases are recorded in Table II.

Table II. Haemoglobin (Hb) and red cell count (RBC): preoperative values.

Sex	<i>n</i>	Hb g/100 ml	Range	<i>n</i>	RBC mill./mm ³	Range
F	8	12.4 ± 0.7	9.8—14.9	8	4.51 ± 0.1	3.9—5.0
M	30	13.0 ± 0.3	9.6—15.4	29	4.35 ± 0.1	3.5—5.4

WHITE CELL COUNT

Leukocytosis (W.B.C. $> 9,000/\text{mm}^3$) was present in two cases. Both patients had active tuberculous lesions in the pericardium, and one in the lungs as well. In the other cases, the WBC was within normal limits.

Differential count. Eosinophilia (> 12 per cent) was present in two cases, and lymphocytosis (< 52 per cent) in one case.

OTHER VALUES

The serum chlorides, alkali reserve and haematocrit values all lay within the normal range.

2. URINE

Proteinuria of varying degree was present in 13 cases; in two of them the sediment contained formed elements.

No permanent rise in *non-protein nitrogen* was present in any case. Although an initially high non-protein nitrogen level was found in a few cases, repeated determinations showed a return to normal values.

3. LIVER FUNCTION

The enlarged liver of chronic constrictive pericarditis was first observed and placed in its proper relation by PICK. Owing to its apparent similarity to cirrhosis of the liver, it has sometimes been incorrectly diagnosed as Laennec's cirrhosis, with resulting faulty treatment. The enlarged liver, known in France as *cirrrose cardiaque*, has been regarded by many authors to explain the poor final results in some cases (*e. g.* EDWARDS & BARLOW). It has sometimes been treated surgically as Laennec's cirrhosis, without the presence of constrictive pericarditis being recognized (DETERLING & HUMPHREYS).

In order to ascertain the liver function in the present series, determinations were made of the serum protein, serum bilirubin, thymol turbidity, alkaline phosphatase and urinary urobilin. Liver biopsy was performed in three cases, since palpation had shown the liver to be unusually hard and enlarged.

SERUM PROTEIN

The total serum protein was determined in 36 cases, and a fractionated serum protein analysis was performed in 24 cases. The albumin—globulin ratio was $< 1:1$ in 4 patients (Cases 4, 15, 21 and 34), between 1 and 2:1 in 14, and $> 2:1$ in 6 patients (see Table III and Fig. 4).

Although Case 4 received 4,300 ml of serum over a period of 16 days, concurrently with dehydrating treatment, there was no demonstrable

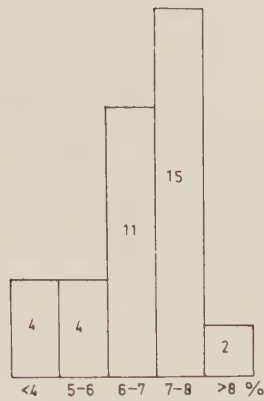


Fig. 4. Serum protein level (%) before operation in the 36 cases in which data were available.

Table III. Total serum protein and fractionated serum protein: preoperative values.

<i>n</i>	Serum protein %	<i>n</i>	Albumin %	Globulin %	A/G
36	6.44 ± 0.21	24	3.94 ± 0.2	2.62 ± 0.2	1.5:1
Range	4—9.26		1.73—5.56	1.17—5.62	

effect on the serum protein. In Case 15, the serum protein was unaffected by blood transfusions, injections of human albumin and dehydration. In Case 21, an exacerbation occurred during oral treatment with an amino acid preparation (Aminosol Vitrum), as well as injections of human albumin, several transfusions of whole blood and one of serum. The oedema increased, but the serum protein underwent no change. Finally, Case 34 received 27 transfusions of serum, 300 ml in each, in the course of 12 weeks, together with dehydrating treatment, but the serum protein remained below the oedema level throughout this period. In this case, a diagnostic error was made, in that the pericardial changes were not believed to be responsible for the liver enlargement and low serum protein. A circulatory study, showing that the blood flow through the liver lay within normal limits, was taken as an indication that other factors were of greater importance for the protein deficiency. The low serum protein persisted unchanged for over seven years, and rose only after venous stasis had been relieved by operation.

SERUM BILIRUBIN

According to White, venous stasis is not the only cause of jaundice, unless it has been present for several years, when it may be associated with some degree of cirrhosis. The factor responsible for the rise in serum bilirubin in heart disease is still uncertain. Some authors have ascribed it to the poor blood flow in the liver, and others to the high pressure in this organ, preventing the free outflow of bile.

The serum bilirubin was determined in 34 cases in the present series. The mean value was 1.10 ± 0.13 mg/100 ml, ranging from 0.20 to 3.0 mg; in five cases it was over 2 mg/100 ml (Table IV).

Table IV. Serum bilirubin, thymol turbidity and alkaline phosphatase: preoperative values.

Test	<i>n</i>	Mean	Range	Upper limit (U1)	> U1
Serum bilirubin mg/100 ml	34	1.10 ± 0.13	0.20—3.0	2.0	5
Thymol turbidity units	25	2.65 ± 0.44	0 — 9.9	6.0	3
Alkaline phosphatase units/100 ml ..	33	11.2 ± 0.9	0 —23.2	10.0	17

TAKATA TEST AND THYMOL TURBIDITY

The Takata precipitation test was made in 8 of the earliest cases, and was positive in two of them.

The thymol turbidity was determined in 25 cases. The mean value was 2.65 ± 0.44 units, ranging from 0 to 9.9 units; in three cases it was over 6 units (Table IV).

ALKALINE PHOSPHATASE

The alkaline phosphatase content of the serum was determined in 33 cases. The mean value was 11.2 ± 0.9 units/100 ml, with a range of 0 to 23.2 units; a value over 10 units/100 ml was recorded in 17 cases (Table IV).

URINARY UROBILIN

The urinary content of urobilin was increased in 11 of the 21 cases in which this test was made.

LIVER BIOPSY

Liver biopsy was performed in three cases. In all three, histologic examination showed the presence of venous stasis. In two of them (Cases 12 and 34), there was a relatively abundant infiltration of minute fat droplets in the liver cells, but the normal structure of the liver was preserved, and no irreversible changes could be seen. In both cases the chronic venous stasis was relatively long-standing, and the liver was found at biopsy to be exceedingly hard. In the remaining patient (Case 19) the salient features were those of venous stasis, with an increase in the round-cell elements around the central vein and the portal region, and a slight increase in connective tissue.

4. VITAL CAPACITY

The vital capacity is reduced in many pathologic conditions involving the respiratory and circulatory system. In the present series, the following factors play some role in this respect: (1) Conditions affecting the lungs, *e. g.* tuberculosis, or asthma with emphysema. (2) A mechanical effect on the space in the thorax, such as ascites with liver enlargement; pleural thickening, which impairs the mobility of the diaphragm and ribs; empyema and fluid in the pleurae. (3) Heart disease, in which stasis with an increase in the blood volume of the pulmonary vessels encroaches on the alveolar space, and reduces its capacity. PEABODY (1916) found that if the vital capacity fell to 40 per cent of normal in heart disease, dyspnoea almost invariably appeared at rest or on the slightest exertion.

The vital capacity was determined in 25 cases in the present series, and was found to be reduced to a mean 62.3 ± 4.2 per cent of the calculated value. A reduction to 40 per cent of normal or less was recorded in three cases; a statement of dyspnoea at rest was found in only one of them, in whom the vital capacity was lowest, *i. e.*, 25.7 per cent of normal. Seven patients had a vital capacity amounting to 70 per cent of normal or above.

RESIDUAL VOLUME RATIO.—This was investigated in 19 patients. Two of them were women, and one had a high ratio (38.7 per cent). Two of the 17 men had a ratio that was greater than normal (38 and 39 per cent, respectively). Naturally, the high residual volume ratio plays the greatest role in those cases in which it is present concurrently with a considerable reduction in vital capacity.

CHAPTER 6

HAEMODYNAMIC STUDIES

1. CIRCULATION TIME

When this series of cases of constrictive pericarditis was started, few simple clinical methods were available for a study of the haemodynamic conditions. In the course of the investigation, measurements of the peripheral venous pressure and the circulation time from the antecubital vein to the tongue were replaced by more exact methods. Thus, cardiac catheterization and improved techniques for roentgenologic examination have been able to give a better conception of the course of events than that provided by earlier methods. For the sake of comparison, however, the relatively inexact method of determining the circulation time has been retained.

This examination was performed preoperatively in 26 cases, and showed the circulation time to be prolonged to a mean 24.2 ± 1.6 seconds (Table V, page 91). The shortest time, 7 seconds, was recorded in a 10-year-old girl, the highest value recorded being 38 seconds. In 22 cases, the circulation time was 17 seconds or more.

2. PRESSURE MEASUREMENTS

The *peripheral venous pressure* was recorded in Cases 1—6, 8, 9 and 14. It was raised in every case, the average value being 20.3 mm Hg. The lowest pressure was 14.3 mm, and the highest 27.2 mm Hg.

In the remaining cases, the pressure was measured with a heart catheter in the right atrium. In Cases 7, 10, 11, 13, 15 and 38, it was recorded with a water manometer, and in the others with a Hansen & Warburg capacitance manometer, or a strain-gauge manometer.

The pressure conditions and the appearance of the pressure curves recorded in this way in constrictive pericarditis have been described by BLOOMFIELD *et al.* (1946), RICHARDS (1947), BURCHELL *et al.* (1948), WHITE *et al.* (1948), ELIASCH *et al.* (1950), HANSEN *et al.* (1951), MCKUSICK (1952), SAWYER *et al.* (1952), SCANNEL *et al.* (1952), YU *et al.* (1953), WILSON *et al.* (1954) and others. The characteristic features are stated to be a W-shaped atrial curve, a ventricular curve with a diastolic dip, and raised pressure in the right side of the heart and the pulmonary artery, as well as in the pulmonary capillaries, as a sign of involvement of the left side.

ATRIAL PRESSURE

With regular heart rhythm, the atrial curve is in the shape of an M or W, and the two peaks giving it this appearance are easily recognized (Fig. 5). The first positive wave, the *a* wave, which is produced by atrial systole, coincides with the P wave on the ECG, and is marked on the ventricular curve by a small, upward deflexion at the end of the diastolic plateau. The pressure in atrium and ventricle approach each other since, in this phase of diastole, they form a common chamber, communicating through the open atrioventricular valves.

The second positive wave, the *c* wave, is due to the rising pressure in the ventricles when they start to contract. The pressure is transmitted to the atrium by regurgitation, or via the closed atrioventricular valves, which bulge into the atria. The *c* wave coincides with opening of the semilunar valves, and is seen on the atrial curve as a small positive wave just after the *a* wave. The *a* and *c* waves are often confluent, so that only one positive wave is visible.

Two factors are considered to be responsible for the negative wave which succeeds the *c* wave. One is the increase in negative pressure in the thorax, owing to ejection of the ventricular contents. The other is the downward movement of the atrioventricular septum caused by ventricular contraction.

The third positive wave, the *v* wave, is a stasis wave. It is due to the inflow of blood from the veins, in which the pressure is high. The negative wave which follows the *v* wave is caused by emptying of the atrium into the ventricle, after opening of the atrioventricular valves. It is accompanied by the diastolic dip and third heart sound characteristic of protodiastolic gallop rhythm. The third sound is caused by vibrations in the wall of the dilated ventricle, due to the impact of blood expelled from the

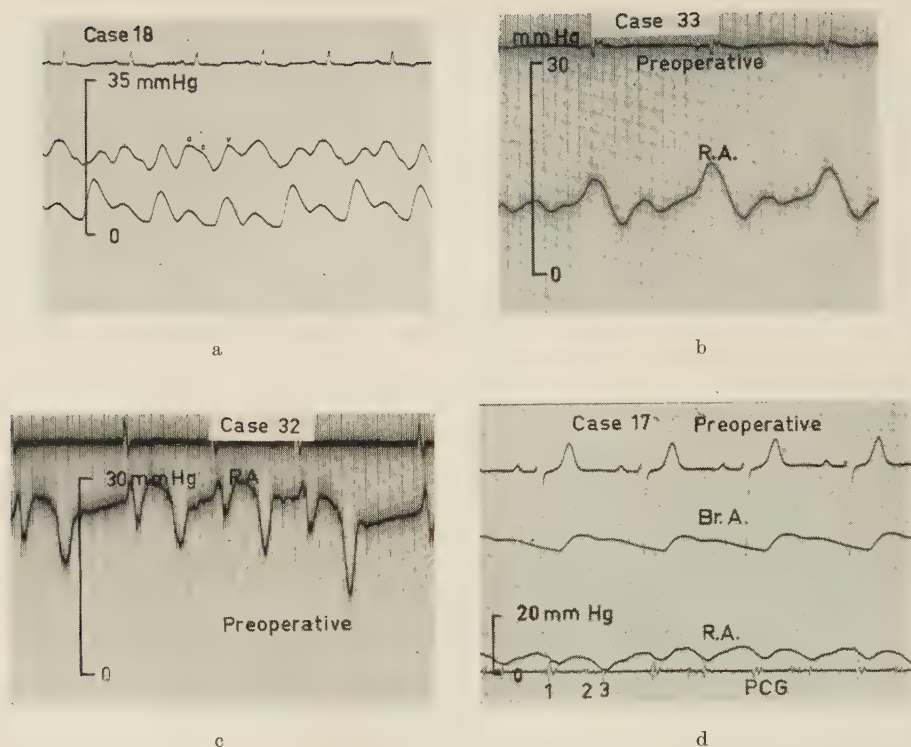
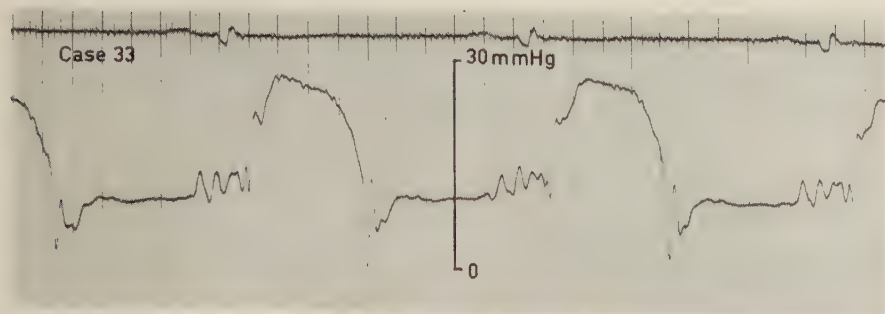


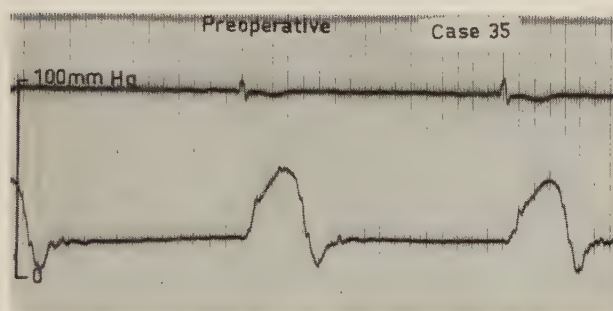
Fig. 5. Right atrial pressure curves in constrictive pericarditis. a. Regular heart rhythm. b. Regular heart rhythm. Type 1 according to Lin & Anache. c. Irregular heart rhythm. Type 3 according to Lin & Anache. d. Regular heart rhythm. Type 6 according to Lin & Anache.

atrium under high pressure (water-hammer effect). Atrial contraction is not apparently of such paramount importance for ejecting the blood into the ventricle, as can be inferred from the ventricular electrokymogram, and from recordings in atrial fibrillation, in which the atrial contractions cannot serve any expulsive purpose.

In the presence of fibrillation, the atrial curve distinctly reflects ventricular action, since in this condition the atria form only part of the reservoir for ventricular filling. Here as well, the curve is W or M-shaped. The *a* wave is naturally lacking, on the grounds of fibrillation, and at the site of the *a* and *c* waves only a positive wave appears, which coincides with the onset of ventricular systole. The subsequent course is similar, with a stasis wave, in which the descending limb represents the rapid filling phase in diastole, and in which the third protodiastolic gallop sound occurs.



a



b

Fig. 6. Typical right ventricular pressure curves in constrictive pericarditis.
a. Regular heart rhythm. b. Atrial fibrillation.

LIN & ANACHE (1956) have classified the atrial curves in constrictive pericarditis into six types, depending on which waves are the most conspicuous (Fig. 5 a—d).

In constrictive pericarditis, the atrial curve is on a higher level than normally. In the present series, the mean value for the mean pressure in the right atrium in 28 cases was 16 ± 1.3 mm Hg (Table V, p. 91). The lowest pressure recorded was 6 mm, and the highest 37 mm Hg. In 27 cases, the pressure exceeded 6 mm Hg.

When the right ventricle is dilated, one of the functions of the pericardium is to prevent relative incompetence of the tricuspid valve; this has been pointed out by BERGLUND *et al.* (1955), among others. In constrictive pericarditis, the pericardial changes imply still greater possibilities of carrying out this function. In tricuspid incompetence, the fall in pressure associated with downward traction of the base of the heart does

not occur. Absence of this fall in pressure was not observed in any of the present cases.

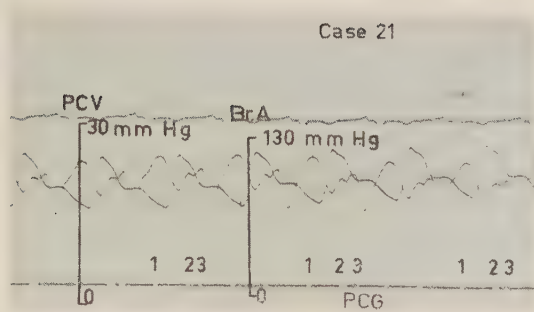
The pressure course in the left atrium is similar to that in the right. In the present series, this was shown by catheterization of the left atrium through the foramen ovale (Fig. 7 b), by direct puncture according to BJÖRK (Fig. 8 b), and by recording the PCV pressure (Fig. 7 a). In Case 21, the pressure was recorded on the same occasion in both atria, with a catheter advanced through the foramen ovale. Figures 7b and 7c show that the curves are not entirely synchronous; since the same catheter was used for both recordings, the time lag could be calculated. The pressure course was found to start 0.011 sec earlier in the right atrium than in the left. Good agreement can be noted between the shape of the atrial and the PCV curve, and the double-peaked appearance characterizes the recordings from both atria, as well as the PCV curve.

VENTRICULAR PRESSURE

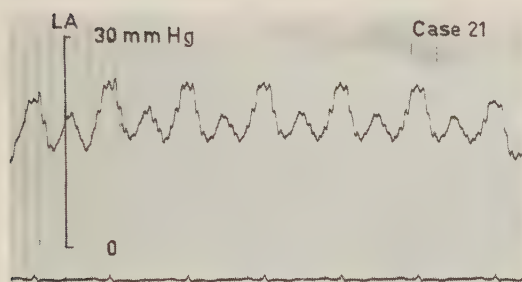
The appearance of the right ventricular curve is highly characteristic. Although it has not been regarded as pathognomonic of constrictive pericarditis, it is of the utmost value as an additional sign to confirm the diagnosis (Fig. 6).

The most characteristic feature is the early diastolic dip. The diastolic dip is also a typical feature of the left ventricular curve, of which the appearance is similar to that of the right ventricle (Fig. 8).

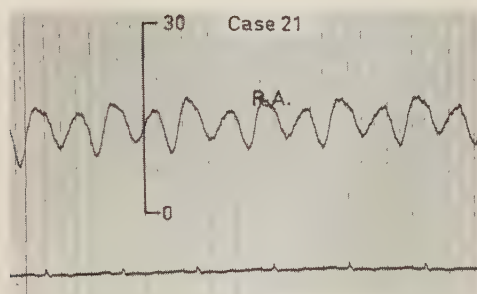
The diastolic dip may, however, be lacking on the right ventricular curve (*e. g.* Case 28), or on the left ventricular curve (*e. g.* Case 34). It is due to the fact that the ventricle, of which the volume is limited by the pericardium, is filled from the atrium and systemic veins, in which the pressure is relatively high. The appearance of this part of the curve consists of a rapid fall almost to zero level, followed by a hasty rise to the level of the atrial pressure. The curve then remains on this level, or diastolic plateau, until the next ventricular contraction. If diastole is sufficiently short, owing to the high pulse rate, this part of the curve may disappear. Vibrations are often observed in the beginning of the diastolic plateau, probably caused by pressure oscillations between atrium and ventricle which, during this phase of the cardiac cycle, form a common chamber with a communication through the open atrioventricular valves. Towards the end of the diastolic plateau, a positive wave is seen; it corresponds to atrial systole. LIN & ANACHE have stated that it is still more accentua-



a



b



c

Fig. 7. Pressure curves in Case 21, showing features typical of constrictive pericarditis. a. PCV pressure. b. Left atrial pressure, obtained by passage of the catheter through the foramen ovale. c. Right atrial pressure.

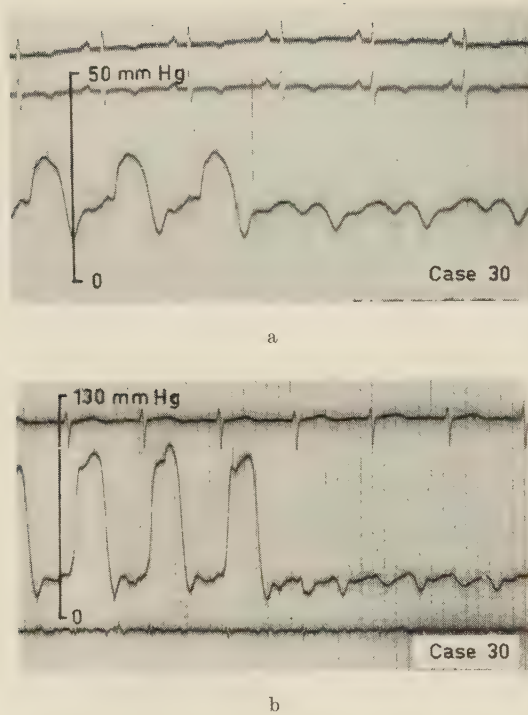


Fig. 8. Pressure curves in Case 30. a. Right ventricular pressure. b. Left ventricular pressure.

ted by the type of atrial curve they have denoted as a giant type of *a* wave. They regarded it as a compensatory mechanism, produced by incomplete filling of the right ventricle in the rapid filling phase. It has already been pointed out that, in chronic constrictive pericarditis, atrial systole probably plays only an inappreciable role in ventricular filling. This pressure wave is due to the fact that, with regular rhythm, atrial systole brings about only an exceedingly slight change in volume in the ventricle, which is already distended to its maximum volume. Consequently, it produces a marked rise in pressure in the ventricle, of which the walls are rigid, owing to the pericardial changes. It is unlikely that this implies any additional filling of the ventricle, as can be confirmed by electrokymography.

The cause of the diastolic dip has given rise to many speculations. ELIASCH *et al.* (1950) have considered that, owing to adhesion of the heart to intrathoracic structures, especially the thoracic wall, a suction effect might arise in the ventricles, when these structures rebound in diastole. The diastolic

dip has, however, been found to persist after these adhesions have been freed, and after decortication, as can be seen on pressure curves recorded at operation. HANSEN *et al.* (1951) have interpreted the diastolic dip as a function of the myocardium. They stated that a pronounced dip to zero level is an indication of good myocardial function, despite constriction, and complete emptying of the ventricle. They regarded these cases to have the best surgical prognosis. If the diastolic dip was less marked, and the rise in pressure occurred at a higher level above the zero line, they considered that some degree of myocardial involvement was to be anticipated. The ventricle then emptied inadequately, and this type of curve was stated to appear in cases of pericarditis with an enlarged heart. Enlargement of the heart is, however, a somewhat inexact term in constrictive pericarditis, since the size of the cardiac silhouette as measured on the roentgenogram does not invariably represent the actual size of the heart.

It might be more appropriate to correlate the dip to the function as expressed in size and emptying conditions on the angiocardiograms. It has generally been easier to perform this study on the right side of the heart than on the left. A dilated right ventricle, with residual blood at the end of systole, indicating impaired myocardial function, was observed fairly often on the angiocardiograms in the present series. Despite this, the diastolic dip was often distinct, with a fall as far as zero level (*e. g.* Case 35). Even if this series is too small to permit any definite conclusions, it nevertheless seems likely that other factors than myocardial function are implicated in producing the appearance of the curve in question. It is evident that the pericardium is of importance in causation of the diastolic dip, since the dip disappears after successful decortication.

The pressure conditions in the right ventricle in constrictive pericarditis have been compared with those in right-sided heart failure, to which it bears a strong clinical and haemodynamic resemblance. Cournand (1945) and others have stated that the systolic pressure in the right ventricle is normal or only slightly raised in constrictive pericarditis, in contrast to right ventricular failure, in which it is high. The diastolic pressure is raised in both conditions. Consequently, in constrictive pericarditis, the pulse pressure is low or approximately normal, whereas it is high in right-sided heart failure. Yu *et al.* focused attention on the diastolic and systolic pressure in the ventricle. They, like Cournand, pointed out that both the end-diastolic and systolic pressure are raised in right ventricular failure. They stated that the ratio of these two raised pressures is always less than 1:3. In constrictive pericarditis, the corresponding ratio is

greater than 1:3 LIN & ANACHE expressed the opinion that, if this ratio is to apply, the systolic ventricular pressure must not exceed 50 mm Hg.

MEAN PRESSURE.—In six cases in the present series, the mean pressure in the right ventricle was measured with a water manometer. It varied between a pressure corresponding to 15 and 26 mm Hg, with an average value of 22 mm Hg.

PULSE PRESSURE.—In 22 cases, the pulse pressure in the right ventricle ranged from 8 to 45 mm Hg (Table V). In the majority of cases, this pressure was normal, but in six cases it exceeded 30 mm Hg. The reason for the normal pulse pressure was that the systolic pressure was relatively less raised than the diastolic. In 10 cases the systolic pressure lay within normal limits, whereas in the others it was raised. The highest value for systolic pressure was 64 mm Hg, and was recorded in two cases. The diastolic pressure was above the normal limit in practically every case. The ratio of end-diastolic to systolic pressure — which, according to YU *et al.*, should be greater than 1:3 — was 1:2.5 in 22 of the present cases.

PULMONARY ARTERY PRESSURE

Constrictive pericarditis has not infrequently been interpreted as mitral disease, owing to the roentgenologic appearance of the heart. The first cardiac catheterizations to be performed served chiefly to elucidate the conditions in the right side of the heart. It was not until this examination showed the pressure to be raised in the pulmonary circulation that it became easier to understand the apparent similarity between the two diseases. The presence of pulmonary hypertension in chronic constrictive pericarditis has been confirmed by the investigations of WHITE *et al.* (1948), ELIASCH *et al.* (1950), BURWELL (1951), MCKUSICK (1952), SAWYER *et al.* (1952) and SCANNEL *et al.* (1952).

The mean pressure in the pulmonary artery was determined in 27 patients in the present series; the mean value was 26.0 ± 1.3 mm Hg, with a range of 17 to 43 mm Hg (Table V). The systolic pressure was recorded in 20 cases, and amounted to a mean 35.6 ± 2.24 mm Hg, ranging from 23 to 60 mm Hg. The corresponding figure for the diastolic pressure was 18.8 ± 1.23 mm Hg (range 9 to 32 mm Hg).

In 19 cases, the pulmonary artery pressure was determined at work.

Table V. Pressure conditions and circulation time: preoperative values.

	Pressure mm Hg				Circ. time sec
	R A	R V Pulse pressure	P A	PCV	
	<i>n</i>	28	22	27	18
At rest					
Mean	16±1.3	—	26±1.3	17.3±1	24.2±1.6
Range	6—37	8—45	17—43	10—26	7—38

Table VI. Pulmonary artery pressure and pulse rate at rest and at work in 19 cases: preoperative values.

	Pressure mm Hg		Pulse/min
	P A		
	<i>n</i>	19	19
At rest			
Mean	27.5 ±1.6	92.2 ±7.2	
Range		52—156	
At work			
Mean	39.9 ±3.2	110.8 ±6.6	
Range		82—155	

In these cases, the mean value for the mean pressure at rest was 27.5 ± 1.6 mm Hg (Table VI). During the first few minutes of moderate work, the pressure rose, and then gradually became stabilized on a slightly lower level than the initial rise. The final determination was made after 10 to 13 minutes' work, and showed the pulmonary artery pressure to have risen to a mean 39.9 ± 3.2 mm Hg (Table VI). In one case, no rise took place in the initially high pressure.

PULMONARY CAPILLARY VENOUS PRESSURE

The raised pulmonary artery pressure at rest, as well as the pathologic rise during light work, are indications of the altered filling conditions in the left side of the heart. These are also reflected in the increased PCV pressure. The PCV pressure curve has been studied by HELLEMS *et al.* (1948). In conformity with their results, BURWELL and SAWYER *et al.* assumed, on the basis of the appearance of the PCV curve, that in constrictive pericarditis it gives an idea of the filling pressure in the left

ventricle, *i. e.*, of the left atrial pressure. In the present series, this could be verified in two cases on curves recorded by means of direct puncture of the left atrium according to Björk, and in one case in which the catheter could be advanced into the left atrium through the foramen ovale. The high PCV pressure is a sign of increased pressure in the left atrium, and thereby of the impediment to adequate dilatation of the left ventricle.

The PCV pressure was recorded in 18 cases. Since it was not my intention to describe the appearance of this curve, only the mean pressure was noted. It amounted to 17.3 ± 1.00 mm Hg, with a range of 10 to 26 mm Hg (Table V).

3. ARTERIAL OXYGEN SATURATION

The oxygen saturation of the arterial blood was determined at rest in 20 cases. It ranged from 88.4 to 100 per cent, with a mean value of 94.1 ± 0.54 per cent. In three cases it lay on the borderline of oxygen unsaturation. The mean value did not differ significantly from that in a normal series investigated at the same laboratory.

In 14 cases the arterial oxygen saturation was studied at work. It was found to vary from 90 to 100 per cent, with a mean value of 95.1 ± 1.0 per cent.

Thus, both at rest and at work, the arterial oxygen saturation was generally within normal limits.

4. OXYGEN UPTAKE

In 21 cases, the oxygen uptake per minute/m² of body surface ranged at rest from 105 to 324 ml.

At work, the oxygen uptake varied between 185 and 465 ml (16 cases). The smallest increase was 53 per cent of the resting value, and the greatest increase 288 per cent.

The ventilation coefficient at rest was 1.9 to 5.09 L/100 ml, and decreased at work to 1.14 to 3.17 L/100 ml.

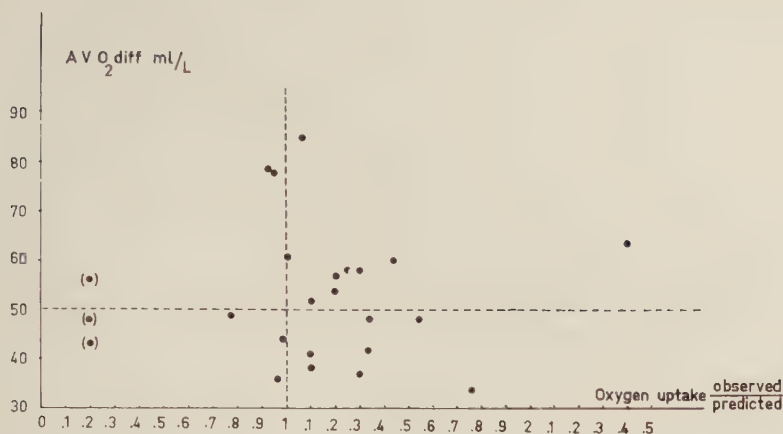


Fig. 9. Arteriovenous oxygen difference (ml/L) at rest in relation to observed/predicted oxygen uptake: before operation. The horizontal broken line marks the upper borderline of the normal arteriovenous oxygen difference, and the vertical broken line the ideal basal conditions. Bracketed dots denote cases in which data regarding the oxygen uptake were lacking.

5. ARTERIOVENOUS OXYGEN DIFFERENCE

The arteriovenous oxygen difference at rest was determined in 24 cases. It was found to range from 33.6 to 85.0 ml/L, and was more than 50 ml/L in 12 cases (Fig. 9).

At work (18 cases) the arteriovenous oxygen difference varied between 48.6 and 143.0 ml/L.

The arteriovenous oxygen difference at work in comparison to that in a normal series can be inferred from Figure 10.

The relation of the arteriovenous oxygen difference to the pulse rate at rest and at work is shown in Figure 11.

6. CARDIAC OUTPUT

The cardiac output in L/min/m² of body surface in relation to the oxygen uptake was within normal limits in a few cases, whereas it was definitely below normal in others (Fig. 12).

Since the main dysfunction in constrictive pericarditis is the low stroke volume, this was determined at rest and at work. In 20 cases, the stroke volume at rest ranged from 25 to 160 ml, with a mean value of 69.0 ± 8.4 ml.

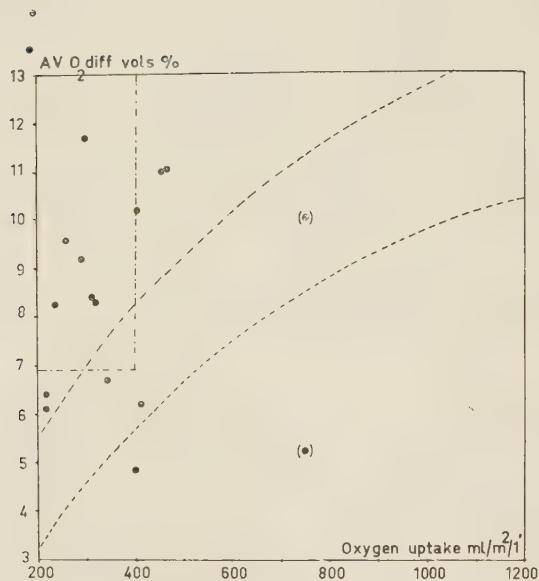


Fig. 10. Arteriovenous oxygen difference (vol. %) at work in relation to oxygen uptake: before operation. The broken lines are the 95 % confidence limits of the regression line best fitting a series of normal subjects according to DONALD *et al.* (1955). The rectangle enclosed by broken and dotted lines is the region of pathologically high A—V oxygen differences at work according to Hickam & Cargill (1948). Bracketed dots denote cases in which data regarding the oxygen uptake were lacking.

At work, the stroke volume varied between 27 and 103 ml, with a mean value of 59.0 ± 7.0 ml. Thus, it underwent little or no change at work.

For the sake of comparison, the stroke volume per m^2 of body surface was calculated. The stroke index at rest was 38.0 ± 4.1 ml (20 cases). This is a significantly lower figure than that given as the normal value for the stroke index. The stroke index at work was 33.0 ± 3.5 ml.

7. ELECTROCARDIOGRAPHY

Immobilization of the heart by adhesions has been considered to produce fixation of the electric axis with changes in position of the body. This has been pointed out by DIEUAIDE (1925), CUSHING & FEIL (1936), HEUER & STEWART (1946) and FRIEDBERG (1950), but has been denied to be of any diagnostic importance by SAMPSON & ROSENBLUM (1934)

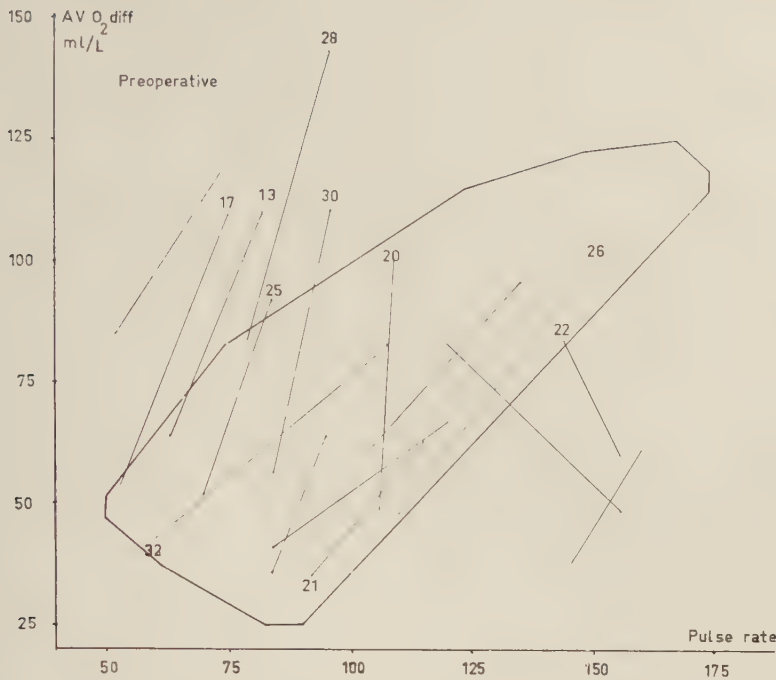


Fig. 11. Arteriovenous oxygen difference (ml/L) at rest and at work in relation to pulse rate: before operation. The region within the thick lines represents the normal variations according to HOLMGREN *et al.* (1957) — (normal series from DONALD *et al.* and FREEDMAN *et al.*).

and FRANCE (1938). Sinus rhythm is more common than fibrillation (HEUER & STEWART 1939, HARRISON & WHITE 1942), and is stated by TUDOR EDWARDS & BARLOW (1946) to occur in 60 per cent of all cases, by WHITE (1951) in two-thirds, and by CHAMBLISS *et al.* (1951) in 69 per cent of all cases.

Enlarged or double-peaked P waves, owing to pressure changes in the atria resulting from increased atrial contraction have been mentioned by CUSHING & FEIL, CASTILLA & AGUIRRE (1938), SINGER-SCHULER (1941), KORTH & WIRKUS (1943), EVANS (1948) and CHAMBLISS *et al.* Changes in the QRS complexes and the T waves have been emphasized by the majority of authors as the most common electrocardiographic signs in constrictive pericarditis. Thus, the reduced amplitude and low voltage of the QRS complexes have been pointed out (CUSHING & FEIL; NOTH & BARNES 1940; HARRISON & WHITE; BARNES & HARRINGTON 1944; HEUER & STEWART; EVANS; VAN NIEUWENHUIZEN 1950; CHAMBLISS *et al.*; WHITE) and the prognosis has then been regarded as poor, owing to greater myo-

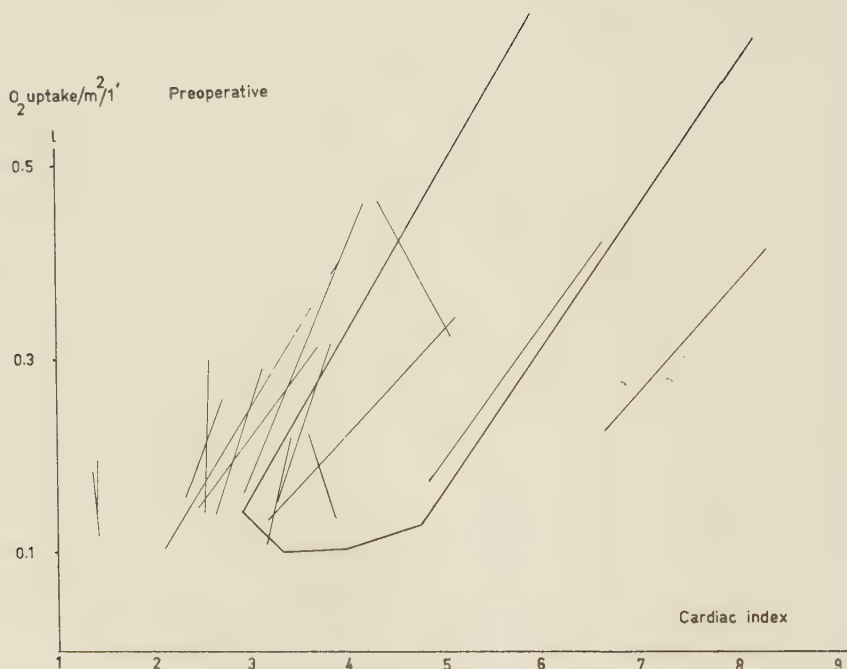


Fig. 12. Cardiac index at rest and at work in relation to oxygen uptake/min/m² of body surface: before operation. The region within the thick lines represents the normal variations according to HOLMGREN *et al.* (1957) — (normal series from DONALD *et al.* and FREEDMAN *et al.*).

cardial damage (KORTH & WIRKUS). In the standard leads, the T waves are isoelectric, flattened, positive or negative in leads I or II or in both (CUSHING & FEIL; NOTH & BARNES; SMITH & WILLIUS; WHITE). Deviation of the S-T segment in the chest leads, in addition to the T wave changes, has been stressed by EVANS and by HARRINGTON.

PRESENT INVESTIGATION

An electrocardiographic examination was made in all the present cases, and the relevant data were available in 37 of them. The most common diagnosis was an impairment of conduction. The rhythm was regular in 24 cases, and probably in an additional case in which reliable data were lacking. Irregular rhythm was present in 13 cases. The conduction time was greater than 0.20 sec in 5 cases, and was within normal limits in 19.

Right axis deviation was present in 4 cases, whereas in 33 cases the electric axis was normally placed.

The QRS interval was normal in all 37 cases. Its voltage was low in 17 cases.

In 10 cases the P waves were double-peaked, and tall or of large amplitude.

The most common changes appeared in the T waves, and were present in 32 cases in the standard leads, in which the T wave was positive in 5 cases and negative in 8. In 5 cases there was a positive T_1 and a negative T_2 and T_3 . In the remaining cases, the T wave was diphasic, isoelectric, and weakly positive or negative.

In the chest leads, which were more or less completely recorded in 32 cases, positive T waves were present in 3 cases. In the remainder, the T waves were isoelectric, diphasic, and weakly positive or negative, as in the standard leads. In the cases with positive T waves in the chest leads, the standard leads showed only inappreciable deviations from normal.

Changes in the S-T interval were present in 12 cases. Since, however, one of the regular effects of digitalis medication is a depression of the S-T segment, the diagnostic importance of this observation should not be overestimated. Depression of the S-T segment unassociated with digitalis medication was found in only two cases.

A tentative diagnosis of constrictive pericarditis was made in the ECG report in four cases, probably after comparison with the other clinical findings.

CHAPTER 7

ROENTGENOLOGIC EXAMINATION

A. Earlier Investigations

In chronic constrictive pericarditis, the roentgenologic changes are partly dependent on immobilization of the heart in the mediastinum, due to the previous infection. ACHELIS (1914) pointed out absence of the descent of the heart in a change from the recumbent to the upright position. ZDANSKY (1931) found it possible to differentiate between accretio and concretio cordis by studying the excursions of the heart and its changes in shape in the lateral view. The limited mobility of the heart in the lateral position and with the respiratory excursions was also demonstrated by FREEDMAN (1937), RIGLER, WANGENSTEEN & FRIEDEL (1941), STEWART, CARTY & SEAL (1943) and GILICK & REYNOLDS (1950). SCHMIEDEN & FISCHER (1926) stated that, at fluoroscopy in the lateral view, the density of the retrosternal space does not decrease if the heart follows with it, owing to fixation to the sternum, which may also be drawn inwards with the action of the heart. Retrocardially, they observed some density due to mediastinal fibrosis. The diaphragm may move irregularly, and be retracted with contraction of the heart (LUKIN 1923).

Fluid in the pleurae, pulmonary stasis and an increase in breadth of the mediastinum, owing to dilatation of the venae cavae, as well as less distinct outlines of the heart, where it is joined to the great vessels, have also been observed (CHAMBLISS *et al.*; FREEDMAN; GILICK & REYNOLDS; STEWART, CARTY & SEAL).

SIZE OF THE HEART

The heart may be small, of normal size, or enlarged (FREEDMAN; MORTENSEN & WARBURG; RIGLER, WANGENSTEEN & FRIEDEL; SCHMIEDEN & FISCHER; STEWART, CARTY & SEAL). An enlarged heart was men-

tioned by LIAWAAG (1947) in 6 of 9 cases, by HARRISON & WHITE in 19 of 37, and by CHAMBLISS *et al.* in 28 of 52 cases. SCHMIEDEN & FISCHER pointed out that, although the heart may be small, its outline on the roentgenogram is enlarged. The size of the heart does not vary in Müller's and Valsalva's experiments.

PERICARDIAL CALCIFICATIONS

It is not uncommon for calcifications, consisting of calcium salts, phosphates and small quantities of carbonate, with traces of soluble sodium salts, to be deposited in the heart or its membranes. This was already described by MORGAGNI in 1762, and later by VARIOT (1898), DIEMER (1899), EWART (1889) and JONES (1901), among others.

Before 1899, it was not considered possible to establish the diagnosis in a living subject. The possibility of visualizing calcifications during life by means of roentgenologic examination was suggested by SIMMONDS in 1908. SCHWARTZ was presumably the first to observe them on the roentgenogram, and in 1911 he reported his second case with calcifications, the patient also having signs of heart failure. Pericardial calcifications were subsequently observed by GROEDEL (1911), RIEDER (1913), WEILL (1916) and MÜLLER (1918).

DIEMER stated that in autopsy material the calcifications were localized to the base of the heart, at the site of the atrioventricular groove, and to the right side of the heart, the parts of the heart in which movement is greatest being uninvolved. Ring-shaped calcifications were described by ROZZA (1898), OBERNDORFFER (1906), LYDTIN (1907), KLASON (1921) and KIRSCHNER & MATTHES (1926), among others. Calcifications are mentioned in all reports of constrictive pericarditis. Thus, HARRISON & WHITE stated that they were present in 16 of 37 cases, PAUL *et al.* in 29 of 53, MORTENSON & WARBURG in 12 of 33, BAILEY *et al.* in 10 to 20 per cent, and McKUSICK in 10 of 20 cases. Calcifications may be present without constriction, as pointed out by DETERLING & HUMPHREYS, GILLICK & REYNOLDS and MATHEWSON.

ROENTGENKYMGRAPHY AND ELECTROKYMGRAPHY

Decreased mobility in the heart region at fluoroscopy has been recognized by most authors, and both quantitative and qualitative changes in the pulsations of the heart have been demonstrated on roentgenkymo-

grams by JOHNSON (1935), STUMPF (1936), BERNER (1939) and RIGLER, WANGENSTEEN & FRIEDEL (1941). The roentgenkymogram shows, under special conditions, the absolute size of the cardiac excursion. In constrictive pericarditis, it is usually smaller than normally, but strikingly large pulsations are sometimes recorded, as an expression of the compensatory increase at those sites in which the movement of the heart is unhampered.

The abnormal pattern of the pulsations appears still more distinctly on the electrokymogram, which permits a more detailed analysis than does the roentgenkymogram. The salient electrokymographic features are the following.

On the ventricular tracings, the filling phase is rapid, and often has a steeper gradient than the emptying phase. The filling phase is ended by an inflexion, and during the rest of diastole has a horizontal course. These electrokymographic changes are an expression of the fact that the ventricle reaches its maximal filling already in protodiastole (GILLICK & REYNOLDS 1950, McKUSICK 1952, HAUBRICH & THURN 1954).

On the aorta and pulmonary artery electrokymograms, a diastolic plateau is present in some cases (HAUBRICH & THURN; McKUSICK). In others, a conspicuous feature is an extra wave, which is synchronous with the end of the rapid filling phase of the ventricle.

When sinus rhythm is present, the most common pathologic finding on the atrial tracings is a prolonged and deepened presystolic deflexion. It is an expression of increased atrial activity during contraction.

None of the foregoing abnormalities on the electrokymogram is pathognomonic of constrictive pericarditis, but pathologic ventricular tracings are strong evidence in favour of this diagnosis. The features in question become pathognomonic only when they occur in combination with calcifications of the pericardium.

ANGIOCARDIOGRAPHY

Introduction by ROBB & STEINBERG (1938), among others, of 70 per cent Diodrast solution as contrast medium brought about a considerable improvement in angiocardigraphic technique. Since then, increasing use has been made of this method for analysis of the conditions in the various chambers of the heart and in the great vessels. It is therefore surprising that the method has not been applied to a greater extent to visualize the

presumptive changes in the caval veins in constrictive pericarditis, instead of fruitless speculations regarding their site and effect.

TUDOR EDWARDS & BARLOW (1946) stated that they had never observed such changes in a series of autopsy cases. SUSSMAN & GRISHMAN (1947) stressed the value of angiocardiology, with which it should be possible to disclose changes, if such had appeared after mediastinitis, which constricted the venae cavae, with resulting high venous pressure. VAN NIEUWENHUIZEN (1950), BAILEY, LACEY & HARRIS (1951) and McKUSICK (1952) were unable to observe any involvement of the caval veins. LYONS, MINNIS & GRIFFIN (1957) reported a case of constriction of the superior vena cava in connexion with constrictive pericarditis, demonstrated by angiocardiology. The caval constriction could be verified at operation, but their report does not make it absolutely clear whether constriction of this vessel was so marked that it played any role in the symptoms.

LEVY *et al.* (1952) suggested the possibility of measuring the thickness of the pericardium by angiocardiology.

B. Present Investigation

Roentgenologic examination of the heart was made more difficult in many cases by the presence of pleural exudate, which masked the cardiac borders. Moreover, measurements of the heart and kymographic studies could be made only after the pleural exudate had been aspirated, or if the examination was made in such a position that the fluid did not interfere with it. Pleural exudate was present in 21 cases, and was bilateral in 14 of them. Four patients had exudate on the left side only, and three on the right side only. Signs of pulmonary stasis were observed in 15 cases.

SIZE OF THE HEART

The heart was enlarged in the majority of cases. In 8 of them, the enlargement was evaluated as being of the same nature as in mitral disease, *i. e.*, it involved the left atrium, and the right atrium and ventricle. In the remaining cases, it was presumably a question of generalized enlargement, and no statement was made of which chambers were affected.

The volume of the heart was recorded in 34 cases. The mean volume

in the 26 males was 614 ± 27 ml/m² of body surface, ranging from 470 to 990 ml/m². Eight of the patients, including two children, were females; the volume ranged from 396 to 716 ml/m² of body surface, with a mean value of 487 ± 38 ml/m².

Only 8 patients had a heart of normal size; 5 were males and 3 females.

PERICARDIAL CALCIFICATIONS

Roentgenologically demonstrable calcifications were present in 22 cases. A survey shows that their most usual site was round the left ventricle. This chamber was entirely surrounded by pericardial calcifications in two cases. In six cases a calcification was present along the border of the left ventricle; it extended onto the posterior aspect of this chamber and continued towards the right ventricle. Corresponding deposits, but chiefly on the anterior aspect of the heart, were observed in one case.

Five cases afforded a good illustration of the classical term "Panzerherz", since the whole pericardium was covered by calcifications. In two cases the right ventricle was surrounded by calcium deposits. In another two, the calcifications were in the form of a band round the ventricular region, leaving the apex more or less free, *i. e.*, they were in the form of a cuff, which surrounded the atrioventricular region and extended onto that of the ventricle.

In one case the calcifications were mainly confined to the apex. In another two they were scattered over the whole pericardium. In one case the calcium deposits lay on the anterior aspect of the heart, on the borderline between the right and left ventricle.

In Case 13, the development of the calcifications could be followed. Roentgenograms taken before onset of the disease showed no signs of calcium deposits. In the course of slightly over three months, streaks of calcium gradually appeared in the pericardium. Their rapid development illustrates the necessity of early diagnosis and operation, since the presence of calcifications generally makes decortication considerably more difficult.

ROENTGENKYMOGRAPHY AND ELECTROKYMOGRAPHY

In the present series, the pulsations of the heart were studied at fluoroscopy, both by direct inspection and, in some cases, by simultaneous recording of the roentgenkymograms. In other cases, an electrokymo-

graphic examination was made. A common feature of all the cases was that the movements of the heart were evaluated at fluoroscopy as being extremely small.

ROENTGENKYMGRAPHY.—This examination was made in 17 of the earliest cases. In 9 of them the pulsations were exceedingly small along the entire border of the heart. Three patients had a limitation of movement on the right side, four on the left side, and in one case the excursion of the heart was decreased posteriorly. In no case were the movements of the heart completely normal.

ELECTROKYMGRAPHY.—A preoperative examination was made in 18 cases. The recordings were made with the patient in the upright (sitting) position, under the conditions described in detail by RUDHE (1956). All 18 electrokymograms exhibited changes indicating that certain features, which occurred with great regularity, could be regarded as typical of constrictive pericarditis. A more detailed analysis could be made in 16 cases.

ATRIAL ELECTROKYMGRAMS

The atrial tracings were studied in 11 patients with sinus rhythm. In two of them, data on simultaneous recordings from both atria were lacking, owing to difficulty in performing the examination, as a result of the masking exudate or poor definition of the atrial borders. The characteristic changes in the tracings from both the right and left atrium were as follows (*cf.* Figs. 13 and 14).

1. The presystolic wave on the electrokymogram, which corresponds to the inward movement of the atrium during its contraction, was negative. Its amplitude was increased in relation to the deflexion during ventricular diastole. As a result, the endpoint of the presystolic deflexion lay on a lower level than the corresponding element in the diastolic deflexion. This differs appreciably from the normal type of curve as described by several authors (*e. g.* ANDERSSON; ENGSTRÖM *et al.*; LUISADA & FLEISCHER; ÖDMAN; RUDHE).

2. In the right atrium, the average duration of the presystolic segment of the curve was 0.14 sec (range 0.10 to 0.19 sec), and in the left atrium 0.13 sec (range 0.10 to 0.16 sec). It is considered that the duration of this segment should not normally exceed 0.10 sec (ÖDMAN, and others). The



Fig. 13. Right atrial electrokymogram. Phonocardiogram over apex.

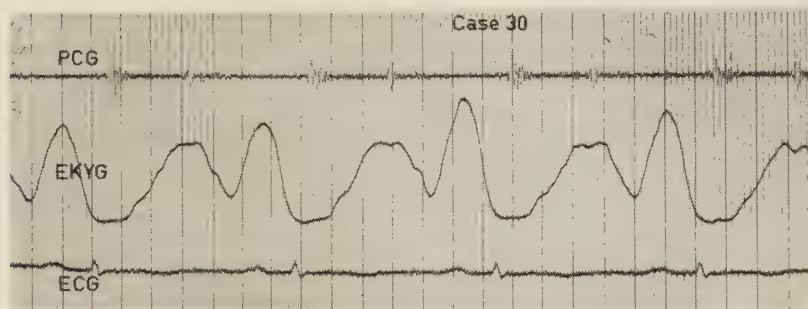


Fig. 14. Electrokymogram from left atrial appendage. Duration of atrial contraction 0.12 sec. Phonocardiogram over apex.

disturbance in activity during atrial contraction, reflected on the electrokymogram, is an expression of the impediment to atrial emptying.

VENTRICULAR ELECTROKYMOGRAMS

The right ventricular electrokymogram was studied in 14 cases; definitely pathologic changes were present in 10 of them (Fig. 15).

The left ventricular electrokymogram was analyzed in 15 cases; unquestionably pathologic features were present on 12 tracings (Fig. 16).

On the tracings from both ventricles, the characteristic change was that the filling phase was more rapid and had a steeper gradient than the emptying phase. After the end of the filling phase, the curve often had a horizontal course during the rest of diastole. In 10 cases, a pathologic electrokymogram was recorded from both the right and left ventricle. In the remaining cases the investigation was incomplete, and could not provide any data on both sides simultaneously. This was due either to difficulties produced by the masking exudate, or to absence of a recording on technical grounds.

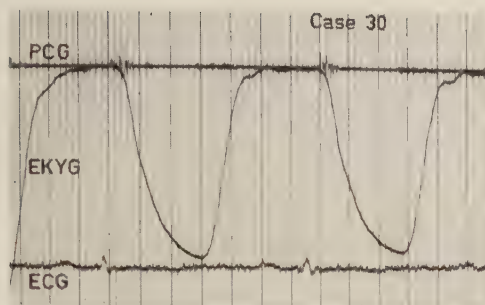


Fig. 15. Right ventricular electrokymogram. Phonocardiogram over apex.

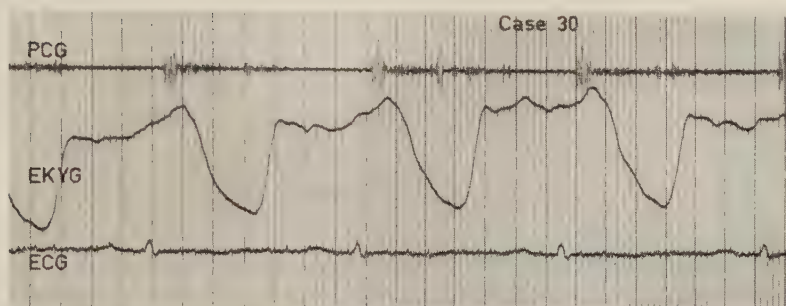


Fig. 16. Left ventricular electrokymogram recorded over apex. Phonocardiogram over apex.

Inverse ventricular pulsations were present in two cases on the left ventricular electrokymogram.

Only one patient had normal pulsations in the segment of the ventricle studied.

COMMENTS

The appearance of the ventricular tracings in the present series is in agreement with the observations made by other workers (*e. g.* GILICK & REYNOLDS; HAUBRICH & THURN; MCKUSICK). The deepened and prolonged atrial contraction in constrictive pericarditis has been described by RUDHE (1954) and by ÖDMAN (1955).

A diagnosis of constrictive pericarditis is not, however, warranted on the basis of the characteristic appearance of the ventricular electrokymograms, since these features may be present in other diseases of the heart and great vessels.

The atrial tracings show that there is an impediment to emptying of both atria. In combination with the raised systemic and PCV pressure, this is good evidence in favour of the supposition that the essential hindrance is to be ascribed to the limited excursion of the ventricles in diastole.

ANGIOCARDIOGRAPHY

Angiocardiographic examination was performed in 26 cases. In the early stages of the present investigation, the technical possibilities of taking acceptable roentgenograms with concurrent injection of contrast medium were limited. Consequently, main attention was focused on visualization of the site of entry of the caval veins into the right atrium, in order to ascertain whether any anatomic changes were present at this site. Considerable advances have subsequently been made in this field of roentgenology. It was therefore possible, in the later part of the investigation, to obtain far more detailed information regarding the appearance of the heart and great vessels in constrictive pericarditis.

Initially, the contrast medium was injected into one of the arm veins, with the patient sitting and unanaesthetized. In recent years, the contrast medium has been injected through a heart catheter, inserted in the superior or inferior vena cava or the pulmonary artery, with the patient under general anaesthesia. Altogether 29 angiocardiographic examinations were made preoperatively in the 26 cases.

The strength of the contrast medium was 70 per cent, except when two examinations were made in relatively close succession, when the concentration was 50 per cent. The quantity injected was calculated in proportion to the body weight, and ranged from 40 to 90 ml.

No complications occurred, either in connexion with anaesthesia, or with injection of the contrast medium.

VENAE CAVAE

On injection into the superior vena cava, the most common observations were that this vessel was wider than normal, that the inferior vena cava became filled by reflux, and that the hepatic veins were also wider than is usual. Corresponding reflux into the superior vena cava was seen when the medium was injected into the inferior vena cava.

In 25 cases in which the caval veins were visualized, no stenosis of these vessels was observed, nor was any present at their site of entry

into the atrium. In most cases the venae cavae were seen to be distinctly dilated.

Dilatation of the superior vena cava, with no or inappreciable enlargement of the heart, is stated to be strongly suspicious of constrictive pericarditis (McKUSICK).

RIGHT ATRIUM

The right atrium was generally dilated, in similarity to the venae cavae. Thus, in the 18 cases in which it was visualized, it was dilated in 15, of normal size in two cases, and possibly slightly dilated in one. No other pathologic changes were observed in this chamber. The contrast medium remained for a fairly long time in the right atrium and venae cavae, as a sign of the prolonged circulation time.

RIGHT VENTRICLE

The right ventricle was enlarged in 12 cases, and of normal size in 6. The variations in volume during ventricular action were seen to range from normal function to small movements, with incomplete emptying of the ventricle and a considerable quantity of residual blood at the end of systole.

PULMONARY ARTERY AND VEINS

The *pulmonary artery* was often somewhat wider than normally, and the circulation through the pulmonary circuit was slow. No stenosis of the pulmonary artery was visible. In the peripheral parts of the lungs, small, irregular vessels of the type occurring in chronic stasis were sometimes observed.

The *pulmonary veins* were also slightly dilated, and in one case (Case 33) it appeared as if two of these veins were stenosed at their site of entry into the left atrium. This observation could not be verified at operation.

LEFT ATRIUM

The left atrium was almost invariably dilated in the 13 cases in which it was sufficiently filled to permit any evaluation.

LEFT VENTRICLE

Owing to dilution of the contrast medium, the conditions in the left ventricle were difficult to judge. This could be done in 9 cases; in one of them the ventricle was larger than normal, whereas in the remaining 8 cases it was of normal size or smaller. The contractility of the left ventricle also ranged from normal to incomplete emptying.

PERICARDIUM

Thickening of the pericardium can also be visualized at angiocardio-graphy. In those cases in which there is an unusually large distance between the contrast-filled cavities of the heart and adjacent structures, *e. g.* the posterior surface of the sternum, the presence of fluid in the pericardium can be suspected. In one patient (Case 12) angiocardio-graphic examination disclosed the existence of a large pericardial exudate.

COMMENTS

The following reflections can be made against the background of the roentgenologic — particularly the angiocardio-graphic — findings. It is reasonable for the atria to become dilated in constrictive pericarditis, owing to the increased venous pressure in both the systemic and pulmonary circuits, which can presumably arise before the pericardial changes have prevented atrial enlargement.

It is also feasible to assume that the working conditions of the left ventricle are less affected than those of the right. Unless myocardial involvement supervenes, the left ventricle works under relatively normal conditions, but cannot receive sufficient blood, since this is prevented by the pericardial lesions. The pericardium sooner or later becomes in-elastic and, irrespective of whether or not any dilatation has previously occurred, it can then take place only within the limits of the constriction. The atria can possibly dilate somewhat in the direction of the pulmonary or caval veins, but any other dilatation must take place towards that part of the heart which works under unaltered conditions.

The right ventricle, which works against the raised pulmonary artery pressure, undergoes greater strain, this being reflected in the dilatation often visible roentgenologically. Consequently, it will, together with the atria, encroach upon the available space, with the result that the ability of the left ventricle to maintain the stroke volume is still further reduced.

The left ventricle's decreased possibility of obtaining blood therefore seems to be an important factor in causation of the symptoms in constrictive pericarditis. The left ventricle is thus small in comparison to the other parts of the heart, as can be verified by angiocardiology. Consequently, an enlarged and dilated left ventricle is presumably a highly unfavourable prognostic sign from the surgical point of view. For, the primary aim of left-sided decortication is to free the left ventricle, thus giving it room to expand freely in diastole.

CHAPTER 8

PREOPERATIVE TREATMENT

The object of preoperative treatment is to decrease cardiac stasis and its results to the greatest possible extent, as well as to correct any nutritional deficiency. Since the cardiac stasis of constrictive pericarditis does not differ markedly from that in any other form of heart disease, it is treated in much the same way. It must, however, be borne in mind that heart failure in the ordinary sense means a failing myocardium, whereas in constrictive pericarditis the symptoms are not, as a rule, attributable to the myocardium, but rather to the hindrance to its efficiency constituted by the pericardium. Consequently, therapy does not invariably produce the desired effect. It cannot be too strongly emphasized that, if preoperative treatment fails to give satisfactory results, it should be discontinued, and operation performed at once, to free the usually adequately functioning myocardium. Excessive use of therapeutic media in those cases in which results are slow in appearing may produce exacerbation, and disturbances in electrolyte balance. This will make it necessary to delay the only truly causal therapy, namely, decortication of the heart.

DIGITALIS MEDICATION

In heart failure, administration of the cardiac glycosides is the only direct means of bringing about an improvement in the mechanical efficiency of the myocardium. The value of digitalis in constrictive pericarditis has been questioned. This drug has been found to decrease the cardiac output in normal dogs (HARRISON & LEONARD) and in normal human subjects (BURWELL, NEIGHBORS & REGAN), and the results of its administration to patients with constrictive pericarditis are not striking. BUR-

WELL & STRAYHORN studied a patient with this disease after digitalis therapy, and found that the drug had reduced the already initially low cardiac output. They ascribed this effect more to a lowering of the pulse rate than to a change in stroke volume. In other words, digitalis counteracts the expedient mechanism for maintaining the minute volume by means of the high pulse rate.

WHITE has stated that digitalis is useless for controlling congestion in constrictive pericarditis, but that it may be of value for decreasing the rapid heart rate if atrial fibrillation is present. LYONS & BURWELL found that digitalis had no effect preoperatively, and that sufficiently large doses reduced the heart rate, with a resulting decrease in minute volume. In their opinion, its only sphere of usefulness is after operation, if dilatation and heart failure are feared.

LEQUIME, COURTOY & KENIS studied a case of constrictive pericarditis before and after injection of digitalis, and were unable to observe any reaction to the drug. They took this as an indication that heart failure was due not to incompetence of the cardiac muscle, but to the effect of the constricting pericardium. HARVEY *et al.* pointed out the value of digitalis therapy as a measure of myocardial function, since improvement produced by it denotes that congestion is to be ascribed not only to the pericardial changes, but also to a limitation of myocardial function.

In the present series, digitalis had been administered preoperatively to 28 of the 38 patients, and in one case quinidine sulphate had been given in an attempt to regulate atrial fibrillation. As a rule, digitalis therapy had been instituted chiefly on the grounds of stasis, and less because of the possible existence of myocardial insufficiency. The effects of the drug were hard to evaluate, but in a few cases it produced a definite exacerbation of the symptoms, which necessitated its discontinuation.

DEHYDRATING THERAPY

The fluid retention due to congestion was combatted chiefly by two measures, *i. e.*, mechanical removal of fluid, and administration of mercurial diuretics.

If there were signs of free fluid in the body cavities, in the form of ascites or pleural effusion, aspiration was performed, in order to decrease the detrimental effect on respiration which appears in both cases. Aspiration, which had usually been carried out over a long period before the

patient was referred for surgical treatment, had only a temporary effect, and had to be repeated to make the patient feel relatively comfortable. This repeated withdrawal of fluid merely led to fresh development of ascites or pleural effusion, with a corresponding loss of protein from the circulation. It had thus been ineffective as a therapeutic measure, but was of great value in preparing the patient for operation.

Mercurial diuretics were administered to hasten excretion of the accumulated fluid through the kidneys. In order to reduce the possibility of fluid retention, a low sodium diet was generally given, and the intake of fluids was limited.

As stated earlier, some patients responded to treatment, and lost weight (*cf.* p. 72); these were patients who were admitted directly for surgical treatment, without any extensive preparation. Others gained weight during preoperative treatment (*cf.* p. 72). They were generally patients referred from other departments, where they had been prepared for operation; they then gained weight under the somewhat less severe regimen of the surgical department. Finally, 17 patients were unaffected by this treatment.

Because of the often extremely low serum protein, a high-protein diet was given, together with transfusions of serum or plasma when necessary. Excretion of the extra fluid through the kidneys was promoted by mercurial diuretics. The effect of this extra administration of protein was never particularly marked (*cf.* p. 79). When the serum protein level was initially low, it remained unchanged; this could be expected in view of the unaltered circulatory conditions.

In those cases in which oedema did not react satisfactorily to the treatment described in the foregoing, and was localized chiefly to the legs, scarification was performed. In two cases, it was possible by this means to evacuate large quantities of fluid resistant to other therapy.

LIVER FUNCTION. — Attempts were made to improve liver function by a plentiful supply of glucose and administration of vitamin B. However, since congestion and malnutrition are probably the chief causes of this impairment of liver function, little can be done until congestion is relieved by operation.

COMMENTS

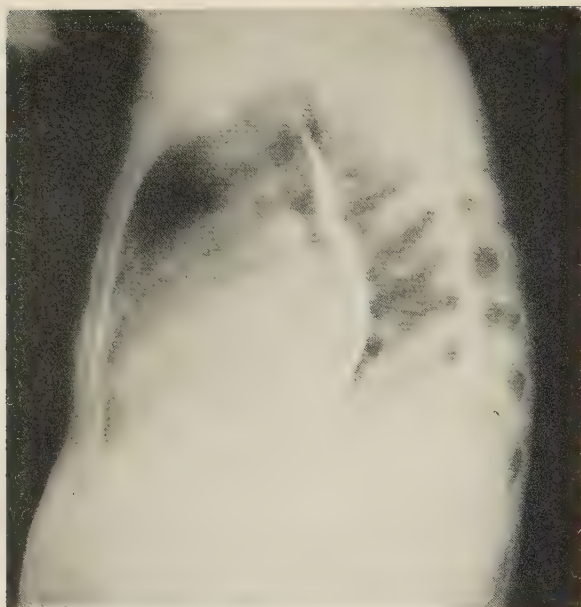
It can be questioned whether the results of dehydrating therapy are of such essential value from the surgical point of view that it is justified to continue it for any length of time. Stationary oedema can scarcely be of decisive importance. If operation is successful, it rapidly subsides. The serum protein is unaffected by extra administration of protein, and rises when the circulatory impediment is removed. The presence of fluid in the abdomen and pleurae considerably hampers respiration, and should be removed by aspiration. But the effect of this intervention is transient, and it should therefore be carried out immediately before operation, to ensure adequate ventilation.

In constrictive pericarditis, venous stasis does not respond to treatment to the same degree as in myocardial insufficiency; one of the main reasons for lengthy preoperative treatment is therefore eliminated. Consequently, it is of the utmost importance to reduce the preoperative period to the minimum beyond the time required for final investigation. Energetic attempts to relieve the symptoms of congestion are more likely to be disadvantageous, unless any severe degree of myocardial involvement is suspected. Even when myocardial disease is present in addition to constriction, the preoperative routine should not be unduly prolonged. Should the myocardial condition also fail to show successive improvement as a result of judicious preoperative therapy, operation should no longer be postponed.

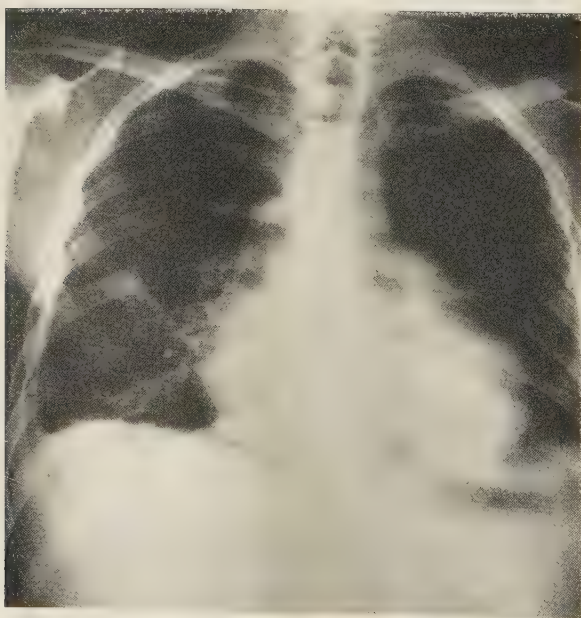
ANTI-INFECTIVE THERAPY

Another exceedingly important preoperative measure is the treatment of infection, particularly tuberculosis. Since a not inappreciable number of cases of chronic pericarditis are caused by tuberculosis, the earlier aim was to produce healing of the tuberculous lesions before operation for pericarditis, since the prognosis was considerably poorer in the presence of active tuberculosis. It was necessary to weigh the severity of active tuberculosis against that of the symptoms produced by constriction. Unless the cardiac symptoms demanded immediate intervention, operation was postponed until tuberculosis was inactive.

The situation is somewhat different nowadays, in view of the anti-biotic and chemotherapeutic agents effective in tuberculosis. Consequently,

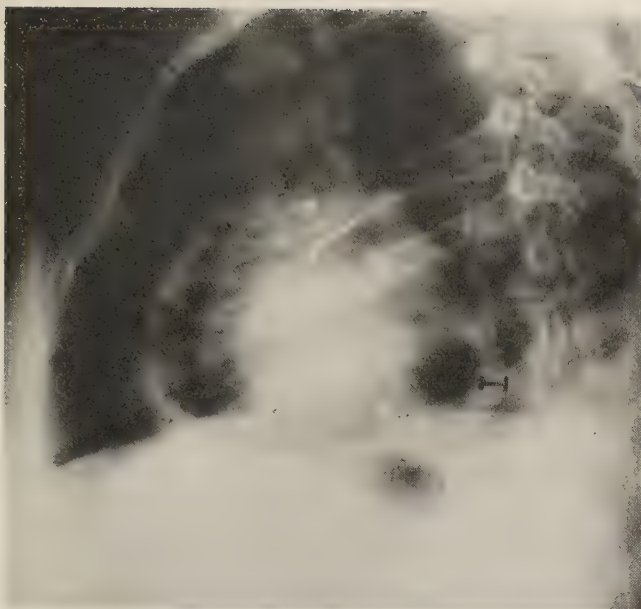


a

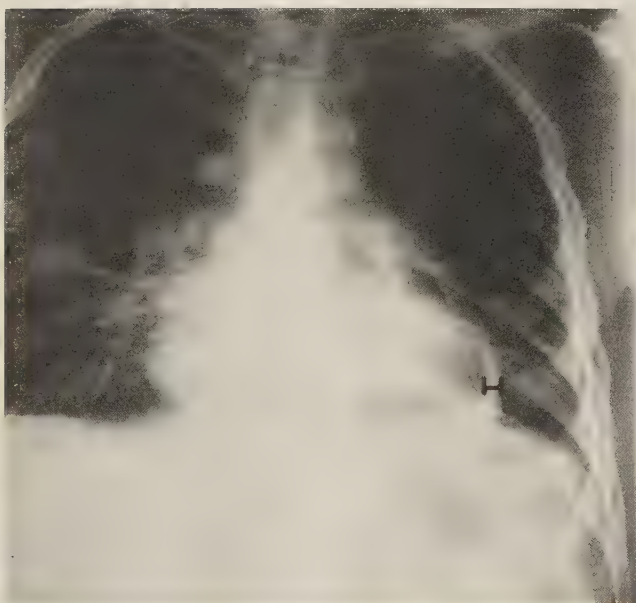


b

Fig. 17 a and b. Outline of the heart before puncture of the pericardium in Case 12 (*cf.* Fig. 18).



a



b

Fig. 18 a and b. Outline of the heart after puncture of the pericardium and injection of air into the pericardial cavity: same case as in Figure 17. Note thickness of pericardium.

there is no longer any hesitation in operating when active tuberculosis is present, provided that antibiotics and chemotherapeutics can be given. Case 37 in the present series illustrates the unfavourable outcome when adequate therapy cannot be applied in tuberculosis (*cf.* p. 53). Cases 14, 20 and 30 illustrate the opposite.

PERICARDIAL PUNCTURE

Pericardial puncture has been an important diagnostic procedure, especially for ruling out the presence of pericardial effusion in a roentgenologically enlarged heart. BECK & MOORE (1925) gave an account of the historical background of surgical drainage for pericardial exudate. As early as 1649, RIOLANUS conceived the idea of draining the pericardium when an inflammatory accumulation of fluid was present. The first successful pericardiotomy was performed by ROMERO in 1819, and the substitution of puncture for drainage was suggested by DIEULAFOY (1873). Many authors have pointed out the risk of injury to the pleura, heart, internal mammary arteries and coronary vessels, as well as various means of avoiding such lesions. Certain advantages are therefore offered by the procedure suggested by REHN, *i. e.*, puncture of the pericardium at a site where both pleura and mammary vessels are lacking, and where the heart is pushed aside by the exudate.

The pericardial exudate, which is replaced by air, may be deceptively like blood, but does not coagulate on aspiration.

The air introduced into the pericardium serves two purposes. Firstly, it replaces the non-compressible fluid, in order to decrease the symptoms of compression. Secondly, it is of diagnostic value to indicate the thickness of the pericardium (Fig. 18). Local application of antibiotics and bacteriologic determination of the organisms present in the pericardial exudate are also of some value.

No complications occurred in connexion with puncture of the pericardium, which was performed in six patients (Cases 9, 12, 18, 20, 32 and 34).

CHAPTER 9

DIFFERENTIAL DIAGNOSIS

In its marked form, constrictive pericarditis is not difficult to diagnose, but no isolated symptom or sign is pathognomonic. Fibrosis of the myocardium is the only disease from which it cannot be distinguished except at operation.

I. Constrictive pericarditis can be differentiated from mitral stenosis on the grounds of the clinical features, the findings at cardiac catheterization in the form of altered pressure and characteristic ventricular pressure curve, presence of pericardial calcifications, and typical ventricular electrokymogram (*cf.* Chapters 4, 6 and 7).

Constrictive pericarditis must be differentiated from the following diseases in addition to mitral stenosis:

II. (a). Tricuspid valve disease, from which it can easily be distinguished by means of angiocardiography and cardiac catheterization.

(b). Cor pulmonale, in which physical examination discloses the cause of heart disease, which can be verified by cardiac catheterization.

III. Other forms of pericardial disease:

(a). Cardiac tamponade; this can be detected by puncture of the pericardium, on roentgenologic suspicions of fluid in the pericardium, which can also be visualized on the angiocardiogram.

(b). So-called mediastino-pericarditis which, actually, need not be distinguished from constrictive pericarditis, since it should be operated on in the same way, if typical symptoms and findings at cardiac catheterization are present.

IV. Extracardiac disease:

(a). Cirrhosis of the liver, in which raised venous pressure is lacking.

(b). Mediastinal tumour in which, although the venous pressure may be raised in the upper part of the body, that in the atrium or lower

part of the body is normal. The tumour can be visualized roentgenologically, and any circulatory impediment at angiocardigraphic examination.

(c). Nutritional oedema, which has been suggested as a differential diagnostic possibility, but which can easily be distinguished from constrictive pericarditis.

CHAPTER 10

SURGICAL TECHNIQUE

Once the diagnosis has been established, operation should be performed in every case. BRAUER's operation, denoted as precardiac thoracotomy, has now fallen into disuse. It is unanimously agreed that constrictive pericarditis should be treated by decortication. Opinions have been at variance only with respect to which parts of the heart are the most important to decorticate, as well as to the details of the relevant surgical technique. From the view now held chiefly by HOLMAN that it is of prime importance to free the right side of the heart, except for the ventricular region, most surgeons have shifted their interest to the left side of the heart.

Two approaches are available for decortication of the heart. One is the "classical" extrapleural pericardectomy according to REHN and VOLHARD & SCHMIEDEN, and the other is the transpleural approach.

A. Extrapleural Pericardectomy

INCISION AND DISSECTION

Extrapleural pericardectomy was elaborated in 1918 by SCHMIEDEN, together with VOLHARD, using KOCHER's flap incision. This is a flap incision through the whole cross-section of the thoracic wall, and includes the third to fifth or sixth ribs on the left side, with the pedicle of the flap on the left. The sternal ends of the third to fifth (or sixth) ribs, as well as the left margin of the sternum, are exposed through this incision. The costal cartilage and a few centimetres of each rib are excised subperiosteally, and the left margin of the sternum nibbled away. The mammary vessels are divided, after ligating them in the upper and lower part of the incision.

When the muscle flap is turned aside, the anterior aspect of the pericardium is exposed. The left pleura is then freed from the pericardium. This step has sometimes led to rupture of the pleura and pneumothorax. The pericardium is incised over the strongly muscled left ventricle and, in the appropriate layer, the heart is freed from the pericardium by both blunt and sharp dissection. The freed anterior layer of the pericardium is excised, and the intercostal muscles and periosteum sutured to its edges. After removal of the part of the pectoralis muscle within the window made in the thoracic wall, skin and subcutaneous fat are sewn over the heart, after inserting a drain in the lower part of the wound.

The possibility of resecting any large portion of the pericardium through KOCHER's incision is fairly limited. In fact, it has generally been possible to remove at most the pericardium over the whole anterior aspect of the heart. Attempts to reach more lateral parts may imply the risk of opening both pleurae, which was earlier regarded as a serious complication. In order to permit a better approach to both sides of the heart, BECK suggested an H-shaped incision, with the two uprights at the third interspace and the junction of the manubrium with the sternum, respectively.

KOCHER's flap incision has been used by many surgeons in extrapleural pericardectomy (*e. g.* BIGGER; CHURCHILL; DECKER; HARRINGTON; HEUER & STEWART; LJUNGDAHL & TENGVALL; SÖDERLUND). It was modified by PIERSOL *et al.*, who trephined the sternum at the base of the xiphoid process, and nibbled away the sternum on the left, together with the third to sixth costal cartilages, and by EAST & HUNTER, who included the sternum in the flap. BLALOCK excised the periosteum and perichondrium together with the costal cartilage, to avoid a chronically suppurating fistula if tuberculosis was the underlying cause. JUZBASIC sutured the bundles of intercostal muscle fibres at the site of the flap incision to the myocardium, in the hope of improving the circulation through newly formed vascular anastomoses. In order to provide better access, LILIEN-THAL and HOLMAN suggested a median sterniotomy according to DUVAL-BARASTY. HOLMAN nevertheless cautioned against using a Lebsche knife for splitting the sternum, since this instrument is liable to produce shock. REHN recommended splitting the sternum from the base of the xiphoid process to the level of the third left interspace.

EXTENT OF DECORTICATION

Following REHN's successful operation, there has been much discussion regarding the extent to which the pericardium should be removed, and also at what sites.

SCHMIEDEN warned against excision of the thickened pericardium over the right side of the heart, since this might lead to dilatation and valvular incompetence. He did not isolate the left phrenic nerve, which is difficult to identify in the scar tissue, but divided it in connexion with excision of the pericardium. He considered that it would imply less work for the heart if it had to pull on the relaxed diaphragm. SCHMIEDEN recommended that excision of the pericardium be started on the left side, since the circulation is maintained chiefly from this part. He regarded decortication of the right side of the heart but not of the left as injurious and, in fact, out of the question. SCHMIEDEN confined pericardectomy to the ventricular region, and advised freeing the apex and basal region, but cautioned against complete decortication, as being too radical an intervention, tolerated only by a heart with exceedingly strong muscles. The left ventricle was freed upwards as far as the atrioventricular groove, to the right as far as the interventricular groove, and also on the posterior aspect. SCHMIEDEN stated that not only should the outer layer of the pericardium be removed, but the epicardial layer should also be separated from the myocardium, to which the scar is often firmly adherent, and into which it may penetrate deeply. He regarded the risk of injuring the coronary vessels as slight, since the scar tissue can easily be detached from them.

SCHMIEDEN thus confined decortication to the left ventricle, but even splitting of ring-shaped calcifications, or resection of narrow strips of pericardium, has sometimes been found to lead to improvement (*e. g.* EDWARDS). Other surgeons, on the contrary, have performed extensive decortication, including the venae cavae and both ventricles.

Freeing of the caval veins, particularly the inferior vena cava, where the exudate may produce constriction, has been stressed by several authors as an important link in decortication (CHURCHILL; HARRINGTON; HOLMAN, and others). CHURCHILL and HEUER & STEWART, in contrast to SCHMIEDEN, expressed the view that there is a not inappreciable risk of injury to coronary vessels. The risk is especially great in the case of the descending part of the left coronary artery, where the scar is often firmly

adherent. HEUER & STEWART had, in fact, inadvertently excised a coronary vessel together with the pericardium in one case.

Even if BLALOCK stated that he had never observed changes producing obstruction of the caval veins, HOLMAN, as well as HOLMAN & WILLETT and BROCK, strongly advocated decortication of this region. BROCK wrote as follows:

".... The old types of flap exposures or wide resections of the cartilages and ribs are bad because they do not leave the bony cage normal afterwards, and also because they give inadequate exposure. A left posterolateral approach by rib resection is not so satisfactory as a median sterniotomy, which gives excellent exposure of the right atrium and the venae cavae as well as the ventricles. Increasing experience has shown that the formerly accepted teaching that it is neither safe nor necessary to uncover the right atrium and cavae is not correct. It is definitely desirable to have free access to the whole of the right heart, so that it can be uncovered and is under full control if haemorrhage should occur."

According to HOLMAN, the strongly pulsating left ventricle is not as easily constricted as is the right, with its thin vessels and atrial walls. HOLMAN's technique therefore consists of decortication of the whole anterior surface of the heart through a median sterniotomy, continuing so far towards the posterior aspect that the right and left borders of the heart are exposed, as well as downwards, to uncover the apex by excision of the thickened pericardium between the lower cardiac border and the diaphragm. The pericardium on the posterior surface of the heart is not removed, this being considered by HOLMAN as unnecessary. His extrapleural intervention is ended by opening the right pleura, to allow the post-operative exudate to flow down at this site, where it can easily be aspirated by puncture.

Just as REHN had earlier recommended decortication of the right side of the heart, CHURCHILL suggested freeing of the right atrioventricular border. This was also advised by HOLMAN and by HOLMAN & WILLETT, although chiefly on different grounds to those of the aforementioned authors. Other surgeons have not intervened on the atria, but have set the borderline for decortication at the atrioventricular groove (BLALOCK & BURWELL; HEUER & STEWART; HOLMES SELLORS; SCHMIEDEN). Incision of the anterior part of the thorax implies operation with the patient in the dorsal position. To decrease strain on the decorticated parts, and to prevent dilatation, CHURCHILL carried out operation with the patient in a dentist's chair, so that the upper part of the body could be sufficiently raised to diminish the venous reflow to the heart.

On natural grounds, the extrapleural approach was that most commonly used in earlier operations for constrictive pericarditis, since it does not

require any complicated form of anaesthesia. This is because the pericardium is exposed without opening the pleurae, which are packed aside before decortication. The risk of injury to the pleurae during their exposure is, however, great. It was therefore recommended by SAUERBRUCH, by SCHMIEDEN & WESTERMANN and by CHURCHILL, among others, that positive pressure anaesthesia should be available during operation, to overcome the complications which could arise if unilateral or bilateral pneumothorax developed. It also became recognized that decortication could be performed only to a relatively limited extent, since removal of the pericardium had to be confined to its anterior part. Access to any larger portion of the pericardium on the left or right side necessitated a transpleural approach.

B. Transpleural Pericardectomy

GENERAL VIEWPOINTS

Transpleural pericardectomy — suggested by VOLHARD (1918) and then performed by SCHMIEDEN — started to compete in popularity with the extrapleural approach chiefly under the influence of the favourable results of anaesthesia and surgical technique in thoracic surgery. It is true that both sides of the heart cannot be dealt with at the same session, but this is an inappreciable drawback, in view of the rare involvement of the right side (ALEXANDER; BAILEY, LACY & HARRIS; BURWELL; McKUSICK; VAN NIEUWENHUIZEN; SCANNEL, MYERS & FRIEDLICH; TUDOR EDWARDS & BARLOW, among others). Consequently, decortication from the left side, when the whole ventricular region is accessible, is fully adequate.

ALEXANDER, as well as OPPENHEIMER, HITZIG & NEUHOF and WINKELBAUER & SCHUR, recommended starting decortication on the left side, with decortication of the right side, if necessary, at a later session. This was regarded as a better procedure for decortication of the whole heart than total removal of the pericardium at one session, which might lead to dilatation.

BLALOCK (1952) stated that the logical conclusion to be drawn from investigations of the impediment to cardiac function was that both sides should be decorticated. After having used a left parasternal incision, which he found inadequate, and then splitting of the sternum with somewhat

better access, BLALOCK's latest preference has been for a left anterolateral, transpleural incision in the fourth interspace, with division of the fourth and fifth costal cartilages, which gives a good approach to both sides.

LIAWAAG, who reported HOLST's operations, also found the intercostal incision to be superior to the anterior. HOLMES SELLORS has made the left transpleural incision as an anterior incision in the third intercostal space, with division of one or more costal cartilages. A similar incision was reported in the cases studied by CHAMBLISS *et al.*

JOHNSON & KIRBY used an anterior incision passing between the two anterior axillary lines and following the fifth interspace, with bisection of the sternum at this level. MULLER has also bisected the sternum into a superior and an inferior portion. The incision ran towards the sternum, parallel to the fourth right interspace. At the right sternal margin, the incision extended to the third interspace, where the sternum was divided, and was continued to the left as far as was necessary. Both these incisions have the common advantage of combining the good posterior exposure of transpleural incisions with the access to both sides of the heart provided by the extrapleural approach.

The most common left transpleural incision is an anterolatero-posterior incision through one of the interspaces, or through the bed of a resected rib. HOLMES SELLORS has used the seventh or eighth rib, DECKER the sixth left interspace, and CRAFOORD the sixth or seventh rib. For decortication of the right side, the corresponding incision is made through the right pleura.

When the dorsal position has not been necessitated by the anterior incisions, operation has been performed with the patient in the lateral position. The prone position has, however, been recommended by OVERHOLT *et al.*, as being more advantageous for the circulatory conditions with an open thorax in general, and in particular in the presence of constrictive pericarditis.

TECHNIQUE USED IN THE PRESENT SERIES FOR LEFT-SIDED DECORTICATION

The subcostal or transperiosteal incision has a great advantage over other incisions, namely, that it combines good healing conditions and stability in the thorax after operation with a satisfactory approach to the operative field. The healing conditions and stability must be poorer, and

the shock-producing trauma to the thorax far greater, when the sternum is split longitudinally or transversely and both pleurae are opened, as has been recommended. This is especially applicable in view of the fact that many of the patients are elderly.

INCISION AND DISSECTION. — RESECTION

As pointed out by CRAFOORD (1945), the left transpleural lateral incision, preferably at the level of the sixth rib, allows adequate exposure of the whole ventricular portion of the heart, as well as the atrioventricular region on the left side. It is evident from the anatomic conditions and autopsy studies that practically the whole of the pericardium, except for a small part covering the right atrium, can be removed through this incision. The left portion of the inferior vena cava can also be inspected.

Owing to pleural adhesions, it is often necessary to free part of the lung from the pericardium and thoracic wall, in order to reach the heart.

The phrenic nerve runs just below the pleural pericardium, from the base of the heart, slightly in front of the pulmonary hilum, towards the apex. By making parallel incisions on either side of the nerve, from the site where it leaves the mediastinal surface and passes over the pericardium, it can be undermined together with a narrow flap of pleura, and lifted from the pericardium. Although the phrenic nerve is most easily observed in the superior part of the mediastinum, it may be completely embedded in the scar tissue of the pericardium, thus being difficult to identify.

When the phrenic nerve has been freed (Fig. 19), the remaining parts of the pleura covering the pericardium in front of and behind it are dissected; anteriorly, the pleura is freed as far as its line of reflection on the thoracic wall. This is sometimes impossible, owing to complete adhesion of the pleura to the pericardium. Irrespective of whether or not the pleura has been freed, the pericardium is separated anteriorly from the posterior aspect of the sternum, and downwards from the upper surface of the diaphragm. The heart is exposed in this way so that, together with the pericardium, it is freed from its adhesions to the surroundings. In front of the heart, exposure is continued to the pleura on the right side and round the apex of the heart.

When the pericardium has been freed in this way from the surroundings, it is incised from the base of the heart, slightly to the left of the root of the pulmonary artery, over the lateral portion of the left ventricle,



Fig. 19. Freeing of the phrenic nerve and separation of the pericardium from the diaphragm and sternum.

down to the apex (Fig. 20). This incision is continued down to the myocardium, after which the margins of the incision are separated from the underlying tissues sufficiently to allow them to be seized with a forceps. The pericardium is then detached from the myocardium, using sharp or blunt dissection as required. Every effort is made to remove all the layers, so that none remains on the heart and impedes diastolic excursion.

At right angles to the first incision, two incisions are made approximately in the midline. One passes upwards over the left atrium, towards the region of the superior pulmonary vein, and the other forwards and



Fig. 20. Incision through the pericardium at the beginning of decortication. The phrenic nerve is not shown on the drawing.

downwards as a prolongation of the posterior incision (Fig. 20). The four flaps of pericardium thus obtained are used to lift the heart forwards on decortication. The upper posterior flap is freed in the direction of the atrioventricular border on the left side. On dissecting through this border, the thin atrial appendage is often found to be adherent to the inner surface of the pericardium, so that undue traction at decortication may cause it to rupture. The reason for continuing dissection over the atrioventricular border is that calcified remains of exudate are often present in the atrioventricular groove, and cut into it, so that a circulatory impediment resembling mitral stenosis might arise (WHITE).

The upper posterior flap is freed as far as a plane corresponding to the root of the pulmonary artery and the superior pulmonary vein. The order

in which the flaps are freed is immaterial. The lower posterior flap is freed in a similar way over the atrioventricular groove, passing over the posterior part of the atrium to the inferior pulmonary vein, and downwards over the posterior part of the apex, on the postero-inferior aspect of the heart. Dissection of this region is facilitated by traction on this flap and retraction of the diaphragm in the caudal direction. On dissection of the apex and traction, various kinds of cardiac arrhythmia frequently appear (*cf.* p. 134), as well as a transient fall in blood pressure; they cease if dissection is interrupted for a few moments.

The two anterior flaps of pericardium are detached from the heart in the same way as the posterior ones. By displacing the heart slightly backwards, and lifting the flaps upwards, the anterior part of the right ventricle can be freed from pericardium. The borderline of the two ventricles, marked by the anterior descending branch of the left coronary artery, is often adherent to the pericardium. It may therefore be necessary to approach this region from both sides, to avoid injury to the coronary arteries.

At dissection, pericardial calcifications are encountered, which penetrate to a varying depth into the myocardium. Those in the superficial layer can be excised, but those lying more deeply cannot be removed, owing to the risk of perforation. It is, however, rarely necessary to leave any large, confluent calcium deposits in the myocardium, and if calcified rings round any segment of the heart cannot be freed completely, they can generally be divided. It is not uncommon to find encapsulated cavities, which may be remains of the former pericardial space. They may be filled with thick creamy fluid, which is evidently under some pressure, since it usually escapes if a small hole is made.

Large vascular anastomoses between heart and pericardium are seldom present. Oozing of blood from the surface of the heart generally stops spontaneously. Small bleeding vessels can be coagulated by cautery. As a rule, scissors and a scalpel are used for sharp dissection. Blunt dissection can be carried out with a finger. BLALOCK has suggested using a peanut dissector, which is a small swab attached to a forceps. An elevator has been found to be a valuable instrument for dissection; its blunt tip and edge can be used to scrape away the myocardium from the uplifted pericardium.

When dissection has been completed, the four flaps of pericardium remain; they are then excised. They should be retained until the end of decortication, since they are useful for lifting up the heart, and can also

be sutured over any ruptures or haemorrhages of the myocardium (BIGGER). Thus, after dissection, the whole left ventricle, left atrium and, as a rule, the greater part of the right ventricle have been freed. The absence of any changes in the region of the inferior vena cava has also been verified.

DRAINAGE.—After stilling the bleeding in the operative field, one or more drains are inserted to evacuate the postoperative exudate. If the lung has not been fixed to the thoracic wall, a tube in the posterior phrenicocostal sinus will generally suffice. If the lung has been firmly adherent to the surroundings, and has been freed only to the extent required for decortication, it may be necessary to insert a drain in the upper part of the thoracic cavity as well, to prevent pneumothorax on the grounds of leakage of air from the freed lung. Moreover, since it is not possible to introduce a tube into the posterior sinus, which is adherent, it is better to insert a drain in the posterior part of the operative field. The lung will then lie in direct contact with the decorticated heart.

COMMENTS

It has been suggested that the freed flaps of mediastinal pleura covering the pericardium should be spared, and used to cover the heart after decortication. Dissection of these flaps may be difficult, and it is doubtful whether any advantage is implied by such a procedure. Blood and exudate probably collect beneath these flaps, and organization — particularly if tuberculosis is the underlying cause — is apt to lead to fresh constriction. CHURCHILL, who cautioned against subjecting the decorticated heart to an excessive venous reflow, has therefore found the persistence of a small pneumothorax after operation to be no cause of anxiety, since he considered the atmospheric pressure on the heart to be advantageous.

RIGHT-SIDED DECORTICATION

Right-sided decortication is carried out in essentially the same way as that described for left-sided decortication. The phrenic nerve is identified in its course along the venae cavae, in front of the pulmonary hilum. Decortication is then continued by freeing the pericardial flaps, which are resected at the end of decortication. A drain is inserted in the pleura and/or operative wound at the end of operation.

COMMENTS ON CERTAIN DETAILS OF THE OPERATIVE PROCEDURE

In the earliest cases in the present series, determination of the venous pressure was the chief means of establishing the severity of venous stasis. Consequently, it is not surprising that two patients were decorticated from the right side, under the impression that an impediment on this side was mainly responsible for stasis in the inflow tract. In one of them, roentgenkymographic examination undoubtedly contributed to focusing interest on the right side. For, the pulsations along the left border of the heart appeared to be of normal size or larger than normal, whereas pulsations were almost entirely lacking along the right border.

Two patients were operated on through a median sternotomy. In one of them, operation was performed at a hospital in another country, at which this incision was then routinely used in such operations. In the other case, the condition was interpreted as constrictive pericarditis with changes involving both sides of the heart, but more pronounced on the right side. It was therefore hoped to succeed in decorticating both sides concurrently through the median sternotomy.

The latter case illustrates the difficulty of exposing the pericardium without injury to the pleurae. The pleura and pericardium were so firmly adherent in some places owing to the inflammatory changes that, despite cautious dissection on both sides, both pleural sacs were perforated. They had to be sutured, after aspirating the fluid and inflating the lung.

Partly on the grounds of observations at cardiac catheterization, the opinion was gradually formed at our clinic that decortication should be performed at two sessions. At the first session, decortication was to be started on the left side, with removal of as much of the pericardium as was accessible. The right side was to be dealt with, if necessary, at a second session, when the rest of the pericardium would be removed.

Left-sided decortication was performed on 33 patients; in these cases only one operation was carried out. Although both sides were decorticated in one case, sufficient grounds for decortication of the right side were lacking, as could be verified at operation.

THORACOTOMY

MEDIAN STERNIOTOMY.—Median sterniotomy was performed with SCHUMACHER's scissors, the whole length of the sternum being divided. Throughout this procedure, blood invariably oozed from the spongy substance of the sternum.

TRANSPLEURAL APPROACH.—Irrespective of the side operated on, thoracotomy was performed through an S-shaped incision according to CRAFOORD, with resection of one rib from the transverse process up to and including the borderline between bone and cartilage. If a sufficient length of rib is excised, it gives a good view of the whole operative field.

In right-sided thoracotomy, the seventh rib was removed in two cases, and the sixth rib in one. In left-sided thoracotomy, the sixth rib was excised in 22 cases, and in one the incision was made at the lower margin of the sixth rib, which was not removed. In 11 cases the incision was made through the bed of the seventh rib.

PLEURAL ADHESION.—In 16 cases, the space between the parietal and visceral pleura was completely or almost completely obliterated. In these cases, the lung had to be freed from the thoracic wall and pericardium to a sufficient extent to allow exposure of the heart. Dissection of the lung often resulted in slight leakage of air. It was therefore necessary, to prevent pneumothorax, to insert a drain in the highest part of the pleura as well.

PHRENIC NERVE.—The phrenic nerve was always exposed together with the surrounding area of pleura. In one case, however, the nerve could not be identified, owing to the severity of the pericardial changes; it was therefore divided in connexion with pericardectomy.

PERICARDIAL CALCIFICATIONS

As stated earlier, pericardial calcifications were found in more than 50 per cent of the present cases (*cf.* p. 102). Such calcifications have been interpreted as an indication of advanced disease, and have been regarded by many authors as a contraindication to pericardectomy. In INGVAR's and TENGVALL's cases, as in those of MORTENSEN & WARBURG, the results were poorest when calcifications were present. According to SANTY *et al.*,

good results cannot be anticipated in the presence of calcifications in patients over 40 years of age.

The large number of cases with calcifications in the present series made decortication more difficult, as well as more risky. In 16 cases, the calcifications penetrated deeply into the myocardium, with resulting greater difficulty in separating the two layers of pericardium. After unsuccessful attempts to reach them from various directions, there was no other choice than to leave them *in situ*. These calcifications ranged from about 15 to 20 mm in diameter, and were generally isolated phenomena on the surface of the heart. In every case, ring-shaped calcifications round the heart were split, if their localization was such that they could be expected to have a constricting effect on any segment of the heart.

EPICARDIUM. — Removal of myocardial calcifications was not the only difficult problem. In some cases, the epicardium was converted into a thick membrane which, after removal of the outer layers of pericardium, impeded the action of the heart.

In two cases, the greater part of the epicardium had to be left in place. Thus, in Case 12, attempts to detach the adherent epicardium from the ventricles were unsuccessful; it was therefore cut into strips to obviate its constricting effect. Epicardial thickening around the base of the heart, at the level of the atrioventricular groove, was also present in this case, but could be removed by dissection. Similar changes around the basal region of the heart were found in Case 30, but only in a few places was the epicardium so firmly adherent to the myocardium that it had to be left *in situ*. The constricting ring in the atrioventricular groove was less firmly fixed to the underlying structures, and could therefore be removed.

CONSTRICTION SIMULATING MITRAL STENOSIS

Because of the alleged resemblance between mitral stenosis and constrictive pericarditis, attention was focused at operation on observing any pericardial changes that might contribute to this resemblance. Such changes should reasonably be localized to the borderline between the left atrium and ventricle. Calcifications or fibrous bands would then cut into the atrioventricular groove on the left side, so that the mitral orifice would either be prevented from dilating, or would be stenosed by shrinking of the pericardium.

McKUSICK cited five cases of chronic constrictive pericarditis with left atrial enlargement simulating mitral stenosis. WHITE *et al.* stated that constriction of the left ventricle, and calcification in the atrioventricular groove between the left atrium and ventricle, may produce left atrial enlargement similar to that seen in mitral stenosis. The authors reported the necropsy finding in a patient in whom left atrial enlargement was supposed to be due to external pressure on the mitral ring by calcified pericardium.

Enlargement of the left atrium in constrictive pericarditis is obviously an interesting observation, although rarely mentioned in the literature. It is, however, exceedingly difficult to evaluate the functional disturbance on the basis of an autopsy finding, such as that described by WHITE *et al.*

In the present series, calcifications between the left atrium and ventricle were a common finding (13 cases), but only in two cases could the functional effect of this calcification be demonstrated preoperatively, by means of catheterization of the left side of the heart according to BJÖRK. In these two cases, the end-diastolic pressure was determined in both the left atrium and ventricle. In Case 30 no difference was found between these two pressures, but in Case 34 there was a pressure gradient across the mitral orifice corresponding to 12 mm Hg. Because of technical errors, no continuous recording could be made on passage of the catheter from the ventricle to the atrium; the pressures calculated were thus recorded at close intervals, but not concurrently. The latter observation seems to be an argument in favour of the probably very rare possibility that calcification of the atrioventricular groove may constrict the mitral orifice.

At operation, every effort was made to remove or divide ring-shaped deposits between the left atrium and ventricle, in the form of calcified remains of exudate or thickened pericardium. This was to prevent them from producing any rise in filling pressure in the left side of the heart. As far as calcifications and ring-shaped formations localized to the right side of the heart are concerned, it was not intended to remove them at left-sided thoracotomy, since they were inaccessible. Moreover, they were evaluated as being of no importance.

With respect to the ring-shaped constrictions of the atrioventricular groove, pressure recordings made on withdrawal of the catheter from the right ventricle to the atrium never showed any pressure gradient typical of stenosis of the tricuspid orifice. As far as the caval veins are concerned, particularly the inferior vena cava, they could be exposed through the left-sided thoracotomy in some cases. In no case could the inferior

vena cava be observed to be constricted by the pericardium to the extent stated by HOLMAN to be necessary for changes in the blood flow to appear. Nor had preoperative angiocardiology shown the presence of any stenosis in this region. Consequently, no indications existed for decortication of this part of the inferior vena cava.

CARDIAC ARRHYTHMIA

Cardiac arrhythmia is not uncommon in the course of decortication, especially during dissection of the apex of the heart. It is due to two factors, *i. e.*, traction on the flaps of pericardium in order to free parts of the heart that are difficult to approach, and direct irritation of the myocardium.

BECK & MAUTZ (1937) suggested that, when such arrhythmia appeared, a pause should be made in dissection, and a local anaesthetic applied to the surface of the heart. BECK expressed the opinion that such application of local anaesthetic decreased the effect of external stimuli. MAUTZ (1936) recommended preoperative administration of quinidine sulphate to decrease the irritability of the heart.

FEIL & ROSSMAN (1939) and STEWART & BAILEY (1941) made electrocardiographic studies of the cardiac arrhythmias. They were unable to note any decreased incidence when quinidine sulphate had been given preoperatively. The most common changes during pericardectomy were extrasystoles of ventricular or atrial type. Atrial extrasystoles were sometimes succeeded by paroxysmal atrial tachycardia and paroxysms of atrial fibrillation and flutter. Ventricular rhythm sometimes alternated between extrasystoles and paroxysmal tachycardia. Completely normal rhythm was, however, recorded in some cases during the whole operation. Arrhythmia ceased as soon as manipulation of the heart was interrupted. Local application of procaine did not seem to decrease the irritability of the heart but, in the opinion of FEIL & ROSSMAN, it may possibly prevent development of fibrillation. FITZPATRICK *et al.* (1954) studied the effect of procaine applied topically to the pericardium as well as intrapericardially, but were unable to observe any useful effects.

To sum up, it can be stated that interruption of manipulation of the heart seems to be the best way of restoring normal rhythm. Moreover, even if local application of procaine has no apparent effect, it may be useful in preventing conversion to fibrillation. Another important factor, which

is perhaps less often stressed, is adequate oxygenation of the arterial blood, since anoxia greatly increases the irritability of the heart muscle.

In the present series, cardiac arrhythmia was a common occurrence during decortication. It appeared especially on dissection of the pericardium from the apex of the heart, or when particularly strong traction was exerted on the flaps of pericardium, in order to deal with parts of the heart lying behind its border.

Arrhythmia was transient, and generally subsided spontaneously if manipulation of the heart was discontinued for a few moments. If arrhythmia was more conspicuous, which occurred in eight cases, attempts were made to decrease it by local application and/or intravenous injection of 1 per cent procaine solution, warmed to body temperature. Swabs dipped in procaine solution were used to retract the heart on dissection, and the surface of the heart was concurrently irrigated with the same solution.

In Case 15, marked arrhythmia appeared on dissection, with a rise in the heart rate to about 150/minute. Intravenous injection of 1 per cent procaine solution was regarded as having been the most effective counter-measure, but since a pause in dissection was made at the same time, the value of this treatment was hard to evaluate. In Case 24 as well, a rise in pulse rate occurred towards the end of dissection, but it was influenced neither by local nor systemic administration of procaine.

Digitalis was generally administered during decortication of the ventricular region, as a precaution against the risk of dilatation associated with decortication.

PULSE PRESSURE

Since low pulse pressure is one of the symptoms of constrictive pericarditis, a study was made of the extent to which it is influenced by decortication in the course of operation.

In 30 cases, data were available regarding the systolic and diastolic pressure. Three points of time were chosen to give an idea of whether or not any change took place. The initial determination of the blood pressure was made after induction of anaesthesia, before operation was started. The average pulse pressure was then 29 mm Hg, ranging from 5 to 60 mm Hg. The next determination was made in the middle of operation, from the point of view of time. The average pulse pressure was 33 mm Hg,

varying from 10 to 60 mm Hg. The last determination took place at the end of operation, after suture of the skin, and before the patient had woken from the anaesthetic. The average value was then 35 mm Hg, ranging from 5 to 60 mm Hg. The increase in pulse pressure between the first and last determination is almost significant ($1 \text{ per cent} < p < 5 \text{ per cent}$).

Low blood pressure throughout the operation was present in one patient (Case 2). In three cases in which only the systolic pressure was determined, a marked rise took place in two cases, whereas in one the blood pressure lay on the same level during the whole operation.

DURATION OF OPERATION

The duration of operation naturally varied with the degree of difficulty of decortication. The average duration was 3 hours 40 minutes. The longest time, 5 hours 35 minutes, was taken in Case 8, in which the great severity of the pericardial changes, with extensive calcifications, made decortication more than usually arduous. The shortest time, 2 hours 25 minutes, was recorded in Case 33, in which decortication was facilitated by the presence of a plane of cleavage in which the pericardium was easily detached from the underlying tissues.

ADMINISTRATION OF BLOOD AND FLUIDS

After decortication, there is a risk of dilatation of the heart. For this reason, CHURCHILL tried to prevent an excessively large reflow to the heart, and warned against blood transfusions. BLALOCK & BURWELL and HARRINGTON have stated that blood or other fluid should not be given during operation, unless special indications exist.

Both incision and decortication are associated with some loss of blood, which must obviously be replaced. At the beginning of operation, the circulation is adapted to a certain venous pressure to maintain circulation. If the quantity of circulating blood is decreased without a corresponding regulation of venous tonus, the venous filling pressure necessarily falls, and the cardiac output decreases. Administration of blood during this stage merely restores the usual working conditions.

After decortication, the persisting high venous filling pressure implies the possibility of dilatation of the ventricle, which has been deprived of

the supporting as well as constricting pericardium. Dilatation beyond a certain degree produces no improvement in the strength of systole, but is more apt to be detrimental, as KUNO was able to demonstrate. Thus, at the beginning of operation, the engorged veins are not the same ominous sign as after the end of decortication. The degree of venous filling is therefore of value to indicate the need of blood transfusions. Obviously, the blood pressure and pulse are equally important factors in this connexion.

In the present series, an intravenous drip infusion was set up during operation in every case. In the earliest cases, it was started with 1,000 ml of saline solution, which always contained cardiac stimulants of the Coramine and Sympatol type. The method was gradually altered, so that the intravenous drip was used for transfusion of blood alone, only sufficient fluid being passed slowly through it to keep the drip open when blood was not given.

In 32 cases, the quantity of blood given during operation was recorded. It ranged from 3,150 to 300 ml, with an average of 1,024 ml. In 32 cases, data were also available regarding the quantity of fluid administered. The range was 4,200 ml to a few hundred ml, in the latter case only enough to keep the drip open. The average quantity was 1,069 ml.

Some shift can be noted in the relative quantity of blood and fluid administered during operation. In the first 15 cases, an average 800 ml of blood and 1,700 ml of fluid were given, whereas in the last 17 cases, the average quantity of fluid was 400 ml and that of blood 1,200 ml. Thus in the last 17 cases, the quantity of blood was increased by 50 per cent, and the quantity of fluid decreased by 76 per cent.

In the early operations, electrolytes were supplied in the form of physiologic saline. This was subsequently replaced by Ringer's solution. At the time of writing, small quantities of glucose solution are given in between blood transfusions. Macrodex solution was used on a few occasions during a short period, but it was found to have no evident advantage over other fluids used earlier or later.

COMPLICATIONS DURING OPERATION

Serious complications may threaten if the myocardium is so severely involved that it ruptures or is perforated on attempts to free it from the covering pericardium. Both a successful and a fatal outcome have been reported in such cases by SAUERBRUCH and by ZENKER, among others.

The risk of injury to the thin atrial walls has caused several authors to warn against decortication of this region (BLALOCK & BURWELL; CHURCHILL; HEUER & STEWART, and others). BIGGER recommended that the pericardium should be spared until decortication was completed, so that it could be sutured over ruptures of the myocardium, which is generally too friable to be sutured in the ordinary way.

In the present series, haemorrhage from the heart was the most common complication associated with operation. Profuse haemorrhage from the heart occurred in 10 cases during dissection. In 6 of them it arose during dissection of the left atrium, when the atrial appendage lying on the inner aspect of the fibrosed pericardium was injured. Haemorrhage could be arrested in every case by suture of the atrium.

In two cases, haemorrhage was due to injury to the left atrium itself, or to a branch of the coronary artery close to it. In one of these cases, tissue adjacent to the lesion was sewn over it; in the other, a piece of pericardial tissue was stitched over the site of bleeding. In one case, the source of haemorrhage was an anastomosis between the pericardium and a coronary artery; the vessel was sutured. Finally, the haemorrhage arose in one case from the right atrium, which was found at dissection to be severely involved. The bleeding was arrested by suture of the atrium. Haemorrhage during operation was not of such a nature that it could be regarded as responsible for death of any of the patients after operation.

In one case, dissection caused brief cardiac standstill; the heart beats were resumed following cardiac massage. As stated earlier, in one case the phrenic nerve was divided when the pericardium was excised.

To sum up, it can be stated that the complications occurring in the course of operation were of a mild nature, and none of the haemorrhages could be denoted as life-threatening.

MORTALITY

Six patients died in connexion with operation. The postoperative mortality thus amounts to about 16 per cent. The corresponding incidence in 1,027 operated cases of constrictive pericarditis collected from the literature is about 19 per cent (see Table VII). The deaths in the present series were as follows.

Case 2 was given slightly more than 16 litres of fluid (blood and physiologic saline) intravenously in the course of 2 1/2 days, and presum-

Table VII. Results of pericardectomy, collected from the literature.

Author	Number of operations	Mortality	Cured	Improved	Not improved	Late mortality
ADA <i>et al.</i> 1951	2	—	2			
ANDREWS <i>et al.</i> 1948	18	5	2	11		
BECK 1944	46	1	31	2		8
BIGELOW <i>et al.</i> 1957	13	2	11			
BLALOCK & BURWELL 1941	20	8	12			
CARALPS 1949	3	—	3			
CECARELLI 1955	30	9	15	6		
CHAMBLISS <i>et al.</i> 1951	61	11	38	6	2	4
CHURCHILL 1936	10	—	6	3		1
COLE <i>et al.</i> 1941	2	1	1			
CRAFOORD 1957	44	8		31	3	1
DACOSTA 1951	10					
DALTON <i>et al.</i> 1956	61	7	32	14	5	
EDWARDS & BARLOW 1946	20	1	10	2	1	5
FRIEDBERG 1944	1	—	1			
HARRINGTON 1947	34	7	18	5		4
HARRISON & WHITE 1942	28	5	14	3		6
HARTL 1948	8	3	5			
HEUER & ANDREWS 1944	18			14		
HEUER & STEWART 1939	7	—	3	4		
HEUER & STEWART 1939	136	41	51	31		13
HEUER & STEWART 1946	18	—	8	7	3	3
HOLMAN & WILLETT 1949	9	1	5	3		
HUNTER & EAST 1939 ..	3	—	1	2		
INGVAR 1938	5	2	3			(1)
IVANESSEVICH <i>et al.</i> 1945	8	2	3	1		2
KALTMAN <i>et al.</i> 1953	12	2	6	1		
KROOK 1954	11	1	6		2	2
LAWRENCE Jr <i>et al.</i> 1948	7	1	6			
LIIVAAG 1947	7	—	4	1	2	
LLOYD & THOMAS 1944 ..	1	—	1			
MAZEL <i>et al.</i> 1952	47					
McKUSICK 1952	17	2	11	3		
MORTENSEN & WARBURG 1948	20	9	7		2	2
OPPENHEIMER <i>et al.</i> 1941	8	2				
PAPO <i>et al.</i> 1955	38	5	33			
PAUL <i>et al.</i> 1948	42	7	15	11		9
PILCHER 1944	1	—	1			
SANTY <i>et al.</i> 1955	78	16	40	13		6
SCHMIEDEN & WESTER-						
MANN 1937	22	8	6	6		2
SELLORS 1955	54	10	30	14		
SETTERGREN 1951	1	—	1			
SÖDERLUND 1942	5	1	3			1
TENGVAL 1938	7	3	4			
DE VERNEJOUL 1951	6	1	5			
WESTERMANN 1944	54	8	20	15		11
WHITE 1951	42	6	25			10
ZENKER 1950	14	1	6	2		5
ZENKER 1955	69					

ably excreted at most 4 litres. Pulmonary oedema appeared on the second day after operation, and could not be controlled. Death occurred on the same day. The essential cause of pulmonary oedema and the fatal outcome was overloading of the circulation with fluids.

Case 8 tolerated the operation well, without the complications anticipated by the surgeon, in view of the severe involvement of both pericardium and myocardium, which was judged to be on a rheumatic basis. Postoperatively, the patient's condition gradually improved, and the venous pressure fell. He nevertheless died suddenly one month after operation, thus confirming the accuracy of the preoperative prognosis. Autopsy showed the right side of the heart to be encased in a calcified shell, as well as the presence of generalized myocardial lesions.

Case 18 had some cardiac arrhythmia during actual decortication, but it responded to intravenous injection of 1 per cent procaine solution. The patient was in good condition immediately after operation, with no disturbances in blood pressure or pulse. Death occurred suddenly in the afternoon of the day of operation. Ventricular fibrillation may possibly have been the cause.

Case 23 had falling blood pressure after operation, and it did not respond to stimulants. She died on the same day, dilatation of the heart, owing to the poor state of the myocardium, being the cause of death.

Case 24 tolerated the operation well, with no marked rise in temperature. However, ventilatory insufficiency was present, owing to the right lung being embedded in fibrous tissue. There was periodic atelectasis, with impaired ventilation on the left, operated side. Tracheotomy was performed too late, and the patient succumbed to the ventilatory insufficiency on the second day after operation.

Case 38 as well can possibly be denoted as having been misunderstood from the ventilatory point of view. An account of this patient, who died on the day of operation, is given under the heading of complications in connexion with anaesthesia (p. 144).

CHAPTER 11

ANAESTHESIA

GENERAL VIEWPOINTS

According to WHITE (1935), a skilled anaesthetist is one of the foremost prerequisites for successful results of decortication. Thus, almost unintentionally, he expressed the view that it is not the anaesthetic itself that is the most important factor, but rather the way in which anaesthesia is administered.

No ideal anaesthetic exists for operation of constrictive pericarditis, but a large number of possibilities are at hand, some of which may be less suitable in one case than in another. The judicious selection of anaesthetic media and techniques has unquestionably contributed to improving the results of operation.

Actually, there is no essential difference in the anaesthetic technique when using the extrapleural and the transpleural approach. Since in extrapleural decortication the risk of opening the pleurae is considerable, it seems simpler from the point of view of technique to foresee this complication than to take the appropriate measures after its occurrence.

SCHMIEDEN (1926), who performed the first large series of these operations, used local anaesthesia, but stressed that general anaesthesia is preferable. Local anaesthesia was used by BLALOCK & BURWELL (1941), IVANNISSEVITCH & MARTIARENA (1945) and ZENKER (1950). Among those who have used general anaesthesia are HUNTER & EAST (1939), STEWART & HEUER (1939), HARRINGTON (1944), IVANNISSEVITCH & MARTIARENA (1945), CLELAND (1949), ZENKER (1950) and CHAMBLISS *et al.* (1951). CHURCHILL (1936) stated that practically any anaesthetic can be used, provided that the oxygen concentration is sufficient.

TECHNIQUE

Even if the choice of anaesthetic is relatively immaterial, the technique is all the more important. Nowadays, the consensus of opinion is that intubation and artificial respiration are essential when the pleura is opened.

ISAACS, BERGLUND & SARNOFF (1954) discussed the effect of positive pressure breathing in cardiac tamponade, a condition that resembles constrictive pericarditis. The constricting pericardium exerts pressure on the heart that cannot, it is true, be measured by means of any raised intrapericardial pressure, but that is reflected in the raised atrial pressure. The aforementioned authors found that the increased extrapericardial pressure lowered the effective filling pressure, which constituted a substantial circulatory disadvantage. They advised using a technique for artificial respiration which produced as small as possible a rise in the intrapleural mean pressure. For this purpose, they regarded an apparatus with alternating positive and negative pressure to be superior to one with positive pressure during the whole respiratory cycle. Opening of the thorax had a similar effect to that of positive pressure breathing. The severer the degree of tamponade, the greater was the effect of positive pressure breathing.

Even if the two conditions are not identical, it is nevertheless possible to draw some conclusions from the investigation. A respiratory machine set at a known, maximal pressure seems to be better suited for counteracting the unfavourable effect than manually controlled respiration, when the maximal inspiratory pressure varies with too many factors to be estimated. As far as apparatus is concerned, one with negative pressure during expiration is superior to a positive pressure respiratory apparatus.

The pulmonary circulation is naturally another factor influenced by pressure breathing. The greater the inflation of the lungs, the smaller is the volume of blood flowing through them. Consequently, to the already increased work of the right ventricle, due to pulmonary hypertension, is added the work of driving the blood through the vascular bed of one lung, which is reduced by the alveolar pressure. The other lung is more or less compressed when it is packed aside for decortication of the heart. The main task of the anaesthetist is therefore to provide anaesthesia that is satisfactory from the point of view of oxygen supply, with a respiratory mean pressure as low as possible, to avoid interfering with filling of the heart and circulation through the lungs.

In the present series, as stated earlier, all fluid in the abdominal and pleural cavities was withdrawn in connexion with operation, to obviate its detrimental effect on respiration and circulation. The patient was given adequate preoperative medication, which could be supplemented before or during operation, if required. A drip infusion was set up in a suitable vein, for injection of blood, stimulants or anaesthetic medium.

The anaesthetic was given through an endotracheal tube with the help of a spiropulsator, and in the most recently operated cases with an Engström respirator equipped with an aggregate for anaesthesia. The latter procedure permits active expiration by means of negative pressure in this phase of the respiratory cycle. The effect of this measure has not yet been studied in this series, owing to the small number of cases in which it has hitherto been applied.

Data regarding the anaesthetic used were available in 36 cases. In four it consisted of cyclopropane. In 16 cases, an intravenous barbiturate combined with nitrous oxide and oxygen was given, curare being used as a muscular relaxant. In 16 cases, succinylcholine iodide (Celocurin, Vitrum) was used instead of curare. Insufficient depth of anaesthesia produced by nitrous oxide and oxygen necessitated administration of ether in four cases, with Celocurin as the muscular relaxant.

An intravenous barbiturate was used in 31 cases, and the quantity injected was recorded in 29 of them. The average dose during operation was 0.5 g of pentothal sodium in 5 per cent solution. The average dose of curare (16 cases) was 50 mg. In 10 cases, the average quantity of Celocurin administered amounted to 2.17 g.

When nitrous oxide and oxygen were given, anaesthesia was in the first plane of the third stage, whereas cyclopropane anaesthesia required a greater depth to permit satisfactory control of respiration. Tracheotomy was performed after operation in one case; roentgenologic examination had indicated impairment of respiratory function, but the patient had been unable to undergo the planned respiratory functional tests prior to operation.

COMPLICATIONS

Few complications occurred during operation or immediately after it that could be ascribed directly to anaesthesia. The difficulty lies, in fact, in ensuring sufficient oxygenation of a cyanotic patient, and determining

whether cyanosis is dependent on the slow peripheral circulation, rather than on unsaturation of the arterial blood.

No determinations of the oxygen supply were made beyond those customary in connexion with anaesthesia. In only one case is there a possibility that death might have been caused by anoxia during operation. This patient (Case 38) showed signs of oxygen deficiency in the course of operation; periodic improvement of ventilation was therefore necessary, operation being interrupted. Postoperatively, respiration was superficial and snapping, although circulation was adequate. Roentgenologic examination disclosed fluid in the right pleura and atelectasis of the right upper lobe. This, combined with compression of the lung on the left, operated side, created the prerequisites for poor ventilation. The patient had difficulty in waking after operation. After transient improvement, a further exacerbation took place, with accentuation of the earlier type of respiration, and death ensued. The combination of anoxia and carbon dioxide retention presents a picture highly reminiscent of this condition, and the vicious circle can be broken only by sufficient ventilation. Unfortunately, at the time in question, this matter was not fully understood, and the possibilities of providing adequate ventilation were not available.

CHAPTER 12

POSTOPERATIVE TREATMENT AND LATE MORTALITY

TREATMENT

ROENTGENOLOGIC EXAMINATION.—A roentgenologic examination of the lungs was made after operation, in order to ascertain whether any pulmonary complications were present. A large pneumothorax was detected in two cases, in one of them together with atelectasis.

OXYGEN ADMINISTRATION.—Oxygen was given routinely, but continuous administration was seldom required beyond the day of operation and the following day. It was administered through a nasal catheter. If a higher concentration of oxygen in the inspired air was indicated, a tightly fitting mask was used.

INTRAVENOUS INFUSION.—In the earliest cases, the cannula was left in place over the second day after operation. This implied not only a risk of overdosage of intravenous fluid, but also of thrombophlebitis. In the later cases, the cannula was already removed on the day of operation, to prevent either of these events.

Blood transfusions, which have been warned against after operation by many authors, were given if indicated. This applied especially when there was a profuse discharge of exudate through the drainage tube or tubes.

DIGITALIS MEDICATION.—Administration of digitalis was continued post-operatively; it was given to 33 patients in connexion with operation and afterwards. In five cases, no digitalis medication whatsoever was given.

DRAINAGE.—The drainage tube (or tubes) were removed within 3 to 4 days, when the discharge had ceased, owing either to blocking of the tube or decreased production.

The average quantity of exudate evacuated in 29 patients was 1,178 ml. Any fluid remaining after removal of the drain was withdrawn by puncture. An inappreciable quantity of exudate appeared in three cases, and in the remainder the patient either did not survive or the quantity was not recorded.

TEMPERATURE.—The highest temperature after operation was an average 39.2 °C (32 patients). In 27 cases, the patient became afebrile after an average 24 days. In five cases the temperature required a longer time to become normalized. All the patients were afebrile five weeks after operation.

ANTIBIOTICS AND CHEMOTHERAPEUTICS.—Antibiotics were administered routinely, both those active against infections in general, and against tuberculosis in particular. Chemotherapeutics were also given, although to a lesser extent.

Penicillin was the antibiotic most commonly given, *i. e.*, in 33 cases. The next in order was streptomycin (27 cases). With respect to chemotherapeutics, a sulphonamide drug was used in four cases; in one of them it produced generalized dermatitis and therefore had to be discontinued. Neoxin was given in two cases, and PAS in one. Of other antibiotics, Aureomycin was used in two cases, and Chloromycetin, Achromycin, Erythromycin and Terramycin each in one case.

OTHER MEDICATION.—Quinidine sulphate was given postoperatively in three cases, to regularize atrial fibrillation. In two of these cases the arrhythmia appeared some time after operation, whereas in one it was already present prior to operation.

Since in two cases antibiotic therapy did not produce normalization of the temperature within a reasonable period, anticoagulant therapy was instituted, together with continued doses of antibiotics.

COMPLICATIONS

In eight cases the postoperative course differed from the usual one. A slight, transient rise in serum bilirubin was observed in several cases and interpreted as haemolysis, but in one patient (Case 14) jaundice ap-

peared two months after operation, and was interpreted as transfusion hepatitis. Normalization of the raised serum bilirubin and thymol turbidity values occurred after slightly more than two weeks.

Dilatation of the heart appeared after operation in Cases 16 and 25. In Case 16, the postoperative course can be denoted as stormy. The patient had low blood pressure and a high pulse rate, and presented a picture of collapse. As a sign of dilatation, a systolic murmur was audible over the whole heart; it may have been due to relative incompetence of the atrio-ventricular valves. Her condition gradually improved, after energetic treatment with stimulants and digitalization. In Case 25, the clinical picture was characterized by a high pulse rate and low blood pressure, but was never directly alarming. Here as well, the condition responded to treatment. In both cases dilatation of the heart was visible roentgenologically during this period.

As mentioned earlier, two patients were given anticoagulants concurrently with antibiotic therapy. In Case 27, the postoperative febrile reaction was not in any way remarkable, but after just over a week there was an inexplicable rise in temperature. After a change of antibiotic, it showed a tendency to fall, but once again rose in the beginning of the second postoperative week. Anticoagulant therapy, together with antibiotic treatment, produced a lasting fall in temperature in the beginning of the third week.

In Case 35, anticoagulant therapy was instituted on the grounds of bilateral thrombophlebitis in the course of antibiotic therapy. After heparin had been given for about 8 days without any marked effect on the temperature, a cerebral accident occurred, with hemiplegia and aphasia. It was interpreted as a cerebral haemorrhage.

Atrial fibrillation appeared after operation in two patients (Cases 31 and 34). In one of them it made its appearance on the fifth day after operation, and in the other on the thirty-first day. Administration of quinidine sulphate restored normal rhythm in both cases.

Tracheotomy was performed in one case (Case 37). The patient had undergone right-sided lobectomy, with complications. No preoperative tests of pulmonary function could be made, owing to the patient's inability to co-operate. Since the lung regarded as the better one was liable to be the seat of complications, tracheotomy was performed after operation. The patient was given artificial respiration until spontaneous respiration was regarded as satisfactory.

LATE MORTALITY

Thirty-two patients survived the operation and the period of hospitalization associated with it. One death occurred six months after operation.

In Case 37, the constriction was due to tuberculous pericarditis. Although the process in the lungs was active, the prognosis was regarded as extremely poor unless the heart was operated on, in view of the severe effect on circulation. Both operation and the postoperative period, during part of which a respirator was used, were uneventful. When the patient was transferred to a sanatorium, some difficulty arose in treating him with suitable antibiotic and chemotherapeutic agents against tuberculosis. Although circulation was improved, the pulmonary disease progressed, and he died slightly more than six months after operation. A remarkable fact is that, despite the presence of fairly acute pericardial lesions, no complications of the pericardial tuberculosis appeared.

Thus, 31 patients remained for follow-up investigation.

CHAPTER 13

CLINICAL FEATURES

1. GENERAL

None of the 31 patients who survived primarily died during the follow-up period, which ranged from 7 months to 9 years 8 months, with an average of 3 years 7 months.

Active work of a varying degree of strenuousness was resumed in 26 cases. Since the relevant occupation ranged from timberman to office worker, the demands on the physical capacity were highly variable and, therefore, on the circulation as well.

Although resumption of work is no poor criterion of the results of treatment, it provides little information on the extent to which the earlier pathologic features have disappeared. Twenty-five patients felt subjectively healthy. Four felt somewhat improved but, although they were aware of the fact that they would presumably not have survived without operation, they were not entirely satisfied with the final results. Temporary improvement had been noticed by one of them, but the former symptoms had gradually returned. One patient felt definitely worse after operation, and in one case it was difficult to judge the merits of the subjective evaluation.

2. PHYSICAL EXAMINATION

GENERAL CONDITION

A consistent improvement in the general condition had taken place after operation. In only one case could it be denoted as affected (preoperative: 17 cases). In this patient (Case 5), all examinations showed the features

typical of chronic constrictive pericarditis. Marked obesity had developed in one patient (Case 15).

VENOUS STASIS

Distinct engorgement of the peripheral veins, especially those of the neck, was observed only in the aforementioned case (preoperatively: 28 cases), in which the presence of constrictive pericarditis was confirmed by other examinations.

CYANOSIS

Cyanosis was present in three patients (Cases 5, 12 and 16), as compared to 20 cases preoperatively. In Case 5 it was marked; in the other two it was fairly moderate, visible chiefly as slight cyanosis of the lips, somewhat accentuated on exertion. The circulation time was prolonged in Cases 5 and 16. Normal oxygen saturation of the arterial blood was recorded at rest in all three cases.

DYSPNOEA

Dyspnoea at rest was present only in Case 5 (preoperatively: 5 cases).

OEDEMA

Oedema, present in 16 cases before operation, was entirely lacking. This applied even in the patients who were only moderately improved, and in the patient with constriction.

ABDOMINAL EXAMINATION

ENLARGEMENT OF THE LIVER. — In four cases (Cases 5, 12, 25 and 27), the liver was enlarged (preoperatively: 36 cases). All these patients had enlargement of the liver prior to operation. In Case 5, enlargement had increased, which could be expected in view of the other symptoms. In Case 12, the liver was perhaps slightly less enlarged than before operation. In Case 25, the liver was scarcely palpable; it had thus decreased in size. Some other factor than venous stasis may have been responsible for enlargement of the liver in Case 27, in which it was of the same size as before operation. In any event, the low atrial pressure in this case rules out venous stasis as being of any importance. The marked symptoms localized

to the liver region, despite normal values in functional tests, are a possible indication of some degree of cirrhosis. Liver biopsy might be helpful to establish the diagnosis.

ASCITES.—In no case was ascites present at the follow-up examination (preoperatively: 17 cases). The marked, recurrent ascites in Cases 4 and 20 disappeared entirely after operation and no paracentesis had been required, nor had it been required in any other case.

PALPABILITY OF THE SPLEEN.—The spleen was palpable in none of the patients (preoperatively: 2 cases).

PULSE RATE

All the patients were afebrile at the follow-up examination. The mean pulse rate at rest was $72 \pm 1.5/\text{min}$ (range 48 to 90/min). This decrease in pulse rate is significant ($p < 0.1$ per cent).

No special examination was made to determine whether a paradoxical pulse was present, and in none of the cases was there a note of this type of pulse.

PULSE PRESSURE

The lowest pulse pressure was 35 mm Hg and the highest 65 mm Hg, the average value being 53 mm Hg. This implies a rise of 40 per cent above the preoperative value. Low pulse pressure (35 mm Hg) was recorded on one occasion in Case 32; the preoperative value was 25 mm Hg. The rise in pulse pressure after operation is significant ($p < 1$ per cent).

AUSCULTATORY FINDINGS

A murmur was audible in four cases after operation; it was systolic in every instance. In three of them (Cases 3, 7 and 27), it was present before operation, whereas in Case 11 it had developed afterwards. The reason for the murmur in this case is uncertain, but it might have been a pleuro-pericardial-cardiac friction rub.

Protodiastolic triple rhythm (gallop sound) was present only in Case 12 (preoperatively: 17 cases). It was present before operation and persisted after it, despite normalization of both ventricular and atrial pressure. Since the early diastolic dip was still visible on the ventricular pressure

curve, it is possible that the prerequisites for the protodiastolic triple rhythm still existed.

THORACIC WALL

Owing to the way in which operation was performed, *i. e.*, freeing of the heart from its adhesions to the surrounding lung, diaphragm and thoracic wall, systolic retraction of the thoracic wall could not be anticipated after operation. Nor was it observed in any of the present cases at follow-up examination.

SUMMARY

To sum up, it can be stated that the symptoms and signs characteristic of chronic constrictive pericarditis had largely disappeared after operation. In the successful cases, improvement in the general condition, normalization of venous pressure, absence of cyanosis, dyspnoea, ascites and oedema, as well as a decrease in liver enlargement, a lower pulse rate at rest and higher pulse pressure, indicated that decortication had fulfilled its purpose.

CHAPTER 14

LABORATORY STUDIES

1. BLOOD

SEDIMENTATION RATE

The erythrocyte sedimentation rate (S. R.) was determined in 30 cases. The mean value was 11.4 ± 3.1 mm/1 hr, with a range of 1 to 86 mm/1 hr. The S. R. was considerably raised in five cases (Cases 3, 8, 25, 28 and 29). In Case 8, it was determined at close intervals after operation, which explains the high value. Cholecystitis was responsible for the raised S. R. in Case 29; in the other cases its cause is uncertain.

RED CELL COUNT AND HAEMOGLOBIN

Table VIII shows the values for the red cell count (RBC) and haemoglobin (Hb) after operation, as well as the corresponding values both before and after operation in certain cases.

Table VIII. Haemoglobin (Hb) and red cell count (RBC): postoperative values (upper two rows), and the corresponding pre- and postoperative values in 3 women and 23 men (lower four rows).

Sex	n	Hb g/100 ml	Range	RBC mill./mm ³	Range
F	3	13 ± 1.6	11.2—14.1	4.2 ± 0.3	3.6—4.7
M	24	13.9 ± 0.3	10.9—15.7	4.7 ± 0.2	3.9—5.4
F	3	Preop. 12 ± 1.3 Postop. 13 ± 1.6	9.9—14.3 11.2—14.1	4.6 ± 0.2 4.2 ± 0.3	4.5—4.8 3.6—4.7
M	23	Preop. 12.9 ± 0.3 Postop. 13.9 ± 0.3	9.6—15.4 10.9—15.7	4.4 ± 0.1 4.7 ± 0.1	3.5—5.4 3.9—5.4

WHITE CELL COUNT

In 26 cases, the white cell count was within normal limits.

DIFFERENTIAL COUNT.— In 17 cases, the differential count lay within the normal borderlines. Relative lymphocytosis (54 per cent) was present in one case, and eosinophilia (6 per cent) in another.

OTHER VALUES

The serum chlorides and alkali reserve were within normal limits in 23 cases.

The haematocrit value was also within normal limits in 22 cases, the range being 37 to 51 per cent. One patient (Case 28) had an unexpectedly low value, *i. e.*, 26 per cent.

2. URINE

Proteinuria of varying degree was present in two patients (Cases 16 and 34); in both of them it had been present before operation. Nothing pathologic was found in the urinary sediment in either case.

The *non-protein nitrogen* ranged from 20 to 44 mg/100 ml (27 cases).

3. LIVER FUNCTION

SERUM PROTEIN

Determinations of the serum protein were made in 30 cases as a criterion of liver function. The mean value for the total serum protein was 6.73 ± 0.12 per cent, with a range of 5.12 to 8.2 per cent (Table IX). No exceptionally low values were recorded, such as those found preoperatively. Thus, in 3 cases the total serum protein was between 5 and 6 per cent, in 16 cases between 6 and 7 per cent, and in 11 cases 7 per cent or above.

The mean value for the albumin fraction was 4.51 ± 0.11 per cent, and for the globulin fraction 2.19 ± 0.17 per cent (Table IX). The average albumin-globulin ratio was thus 2.1 : 1.

In cases with enlargement of the liver postoperatively, no dysfunction of this organ with respect to serum protein was observed. It has been stressed that a rapid rise in serum protein, when it was exceptionally low prior to operation, is a criterion of successful results.

Table IX. Total serum protein: pre- and postoperative values (both pre- and postoperative values available in 29 cases). Fractionated serum protein: pre- and postoperative values (both pre- and postoperative values available in 11 cases).

	<i>n</i>	Serum protein %	<i>n</i>	Albumin %	Globulin %
Preop. . . .	38	6.39 ± 0.2	26	3.9 ± 0.24	2.57 ± 0.24
	29	6.31 ± 0.11	11	3.59 ± 0.05	2.70 ± 0.4
Postop. . .	30	6.73 ± 0.12	17	4.51 ± 0.11	2.19 ± 0.17
	29	6.74 ± 0.14	11	4.44 ± 0.01	2.26 ± 0.02
			11	1 % < Diff. $p < 5$ %	Diff. $p > 5$ %

In three of the four cases with low serum protein (about 4 per cent) before operation, the reaction was unquestionable. Thus, the serum protein rose to a practically normal level within about a month, *i. e.*, in Case 4 after just over a month, in Case 21 after about three weeks, and in Case 34 after two weeks. Inexplicably, Case 15 reacted more slowly, since the serum protein showed no tendency to rise until four months after operation.

On the whole, the change in serum protein after operation took place as follows. When the total serum protein had been markedly low, it rose to a normal level. The previously decreased albumin fraction also rose, so that the albumin-globulin ratio assumed more normal proportions.

The postoperative rise in the albumin fraction is almost significant (Table IX).

SERUM BILIRUBIN

A rise in serum bilirubin was recorded postoperatively in one patient (Case 25). The liver was palpable, but had decreased in size as compared to before operation. In 28 cases, the mean serum bilirubin value was 0.83 ± 0.08 mg/100 ml, ranging from 0.23 to 2.24 mg/100 ml (Table X).

THYMOL TURBIDITY

The thymol turbidity was determined in 25 cases; the mean value was 2.5 ± 0.2 units, with a range of 0.2 to 10.7 units (Table X). A marked rise was recorded in one case (Case 15). In Case 29, the high preoperative value did not fall; this was possibly due to cholecystitis, which appeared after operation. In the other cases, the values were within normal limits.

Table X. Serum bilirubin, thymol turbidity and alkaline phosphatase: pre- and postoperative values.

Test	n	Mean	Range	Upper limit (U1)	> U1
Serum bilirubin mg/100 ml					
Preop.	34	1.10 ± 0.13	0.20—3.0	2.0	5
Postop.	28	0.83 ± 0.08	0.23—2.24		1
Thymol turbidity units					
Preop.	25	2.65 ± 0.44	0 —9.9	6.0	3
Postop.	25	2.5 ± 0.2	0.2—10.7		2
Alkaline phosphatase units/100 ml					
Preop.	33	11.2 ± 0.9	0 —23.2	10.0	17
Postop.	23	7.2 ± 0.2	4.4—17.7		1

Table XI. Thymol turbidity: pre- and postoperative values in 17 cases. Alkaline phosphatase: pre- and postoperative values in 22 cases.

Test	n	Mean	Diff. p
Thymol turbidity units			
Preop.	17	2.40 ± 0.6	—
Postop.		3.1 ± 0.6	
Alkaline phosphatase units/100 ml			
Preop.	22	10.5 ± 0.28	$p < 1\%$
Postop.		7.3 ± 0.19	

ALKALINE PHOSPHATASE

In 23 cases, the mean alkaline phosphatase content of the serum was 7.2 ± 0.2 units/100 ml, ranging from 4.4 to 17.7 units/100 ml (Table X). A raised value was present only in Case 28. In 22 cases in which this determination was made both pre- and postoperatively, the mean value was 10.5 ± 0.28 units/100 ml before operation, and 7.3 ± 0.19 units/100 ml after it. This decrease in serum alkaline phosphatase is significant ($p < 1$ per cent): see Table XI.

URINARY UROBILIN

Urobilin was excreted in the urine in 5 of the 19 cases in which this determination was made. In three of them, urobilin was also present in the urine before operation; in the other two cases, preoperative data were lacking.

LIVER BIOPSY

Liver biopsy was performed in none of the cases after operation. It might, however, have been justified in Case 27, to ascertain whether the persisting enlargement of the liver and the symptoms from this organ were due to some degree of cirrhosis of the liver, despite normalization of the conditions.

4. VITAL CAPACITY

The vital capacity was determined postoperatively in 26 cases. The mean value was 73.1 ± 3.7 per cent of normal, ranging from 43 per cent of the calculated value to normal (Fig. 21).

In 19 cases investigated both before and after operation, the mean vital capacity was 63.5 ± 4.8 per cent of normal before operation, and 72.1 ± 5 per cent after it. In six patients (Cases 5, 7, 17, 22, 25 and 27) the ratio vital capacity/calculated vital capacity had decreased in comparison to the preoperative value. The average decrease in these cases was 14.1 per cent, ranging from 5.7 to 24 per cent. In Case 5 the decrease was due to constriction. In Cases 7, 17, 22 and 25, the postoperative scar tissue and adhesion of the diaphragm contributed to this decrease. In Case 27, asthmatic pulmonary lesions and a greatly increased degree of emphysema were additional contributory factors.

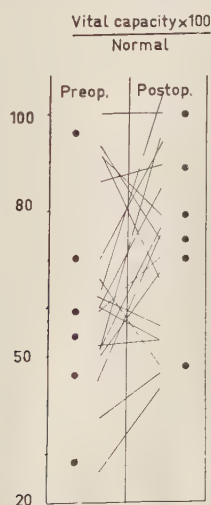


Fig. 21. Vital capacity in per cent of normal before and after operation.

In 11 cases the vital capacity ratio was greater after operation. The average increase was 22.5 per cent, ranging from 3.3 to 46.5 per cent. In two cases the vital capacity underwent no change after operation.

The ratio of residual to total capacity was determined both before and after operation in 16 cases. Some difference could be noted in four of them. One was the aforementioned patient with asthmatic pulmonary lesions. In two patients (Cases 30 and 32) the normal ratio rose to a pathologic value after operation. In one (Case 31) the ratio fell; it was pathologic prior to operation.

To sum up, it can be stated that an increase in vital capacity should appear after operation, in those cases in which no recurrence takes place. This is because the factors contributing to a decrease in vital capacity have been eliminated, *e. g.* ascites, pleural effusion and pulmonary stasis. It is nevertheless evident that this potential increase is counteracted by the formation of postoperative exudate, with organization and conversion into scar tissue. This can be observed most distinctly if decortication has been performed on both sides (*e. g.* Case 12).

CHAPTER 15

HAEMODYNAMIC STUDIES

1. CIRCULATION TIME

The circulation time was determined postoperatively in 26 cases. It amounted to a mean 14 ± 0.7 seconds, ranging from 6 to 20 seconds (Table XII). In 19 cases, both preoperative and postoperative data were available. Before operation, the circulation time in these 19 cases was prolonged to a mean 23 ± 2 seconds; after operation it was a mean 14 ± 1 seconds.

A longer circulation time than before operation was recorded postoperatively in two patients (Cases 3 and 5). At the first examination, Case 3 was 10 years old; at the follow-up examination she was almost full-grown. In Case 5, marked symptoms and signs of constriction had reappeared. In the remaining cases, a distinct decrease in the circulation time had generally occurred, associated with an improvement in circulatory conditions.

2. PRESSURE MEASUREMENTS

The *peripheral venous pressure* was determined after operation in two patients (Cases 8 and 14). The former patient died one month after operation, but the venous pressure, which had been raised before operation, fell practically to a normal level after it. In Case 14, the peripheral venous pressure was greatly raised before operation; at follow-up examination three years later it had fallen to a normal level.

ATRIAL PRESSURE

Cardiac catheterization was performed after operation in 28 cases; the appearance of the atrial curve could be analyzed in 27 of them.

In one patient (Case 5), the atrial curve still had the typical W or M

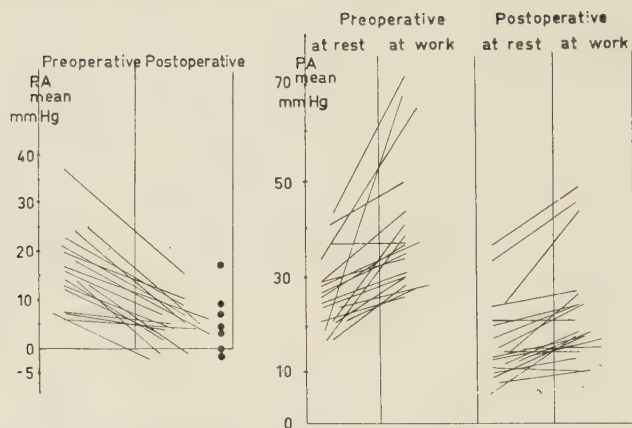


Fig. 22. Mean pressure in the right atrium at rest, and in the pulmonary artery at rest and at work: before and after operation.



Fig. 23. Pressure curves in Case 5, with all symptoms and signs of constrictive pericarditis at follow-up examination.
a. Atrial pressure. b. Ventricular pressure.

Table XII. Pressure conditions and circulation time: pre- and post-operative values.

At rest	Pressure mm Hg				Circ. time sec
	R A M	R V Pulse pressure	P A M	PCV M	
<i>n</i>	28	22	27	18	27
Preop.	16 ± 1.3	8—45	26 ± 1.3	17.3 ± 1	24.2 ± 1.6
<i>n</i>	28	24	27	19	26
Postop.	5.2 ± 0.3	10—42	17.3 ± 1.6	7.6 ± 1.2	14 ± 0.7

Table XIII. Pulmonary artery pressure at work: pre- and postoperative values.

At work	Pressure mm Hg P A M
Preop. (19 cases)	39.9 ± 3.2
Postop. (23 cases)	22.8 ± 2.3

shape (Fig. 23 a); this could be expected in view of the persisting symptoms and signs of constriction.

Since the normal atrial pressure curve is somewhat peaked, it may have an M-like appearance. Consequently, it was difficult in the present series to distinguish the pathologic curves from the normal ones. If a comparison is made between the pre- and postoperative curves, nine of them show a certain similarity, in that peaks are present in both, although they are less distinct on the postoperative tracing. This characteristic feature was lacking in 17 cases, and the atrial curve could therefore be denoted as relatively normal. In two cases in addition to the patient with persisting constriction, the tracings could be denoted as pathologically M-shaped.

The mean value for the mean pressure in the right atrium was 5.2 ± 0.26 mm Hg, with a range of —2 to 17 mm Hg. In 20 cases, the mean pressure in the right atrium was recorded both before and after operation. As a result of decortication, the raised mean atrial pressure of 15.6 ± 1.7 mm Hg was reduced to a mean 4.8 ± 0.27 mm Hg. In 19 cases the pressure was below 6 mm Hg.

The most important result of operation is not, in fact, a return of the atrial pressure curve to a normal appearance, but rather a fall in pressure in the atrium (Fig. 22 and Table XII).

VENTRICULAR PRESSURE

The appearance of the right ventricular curve has been regarded as a typical although not pathognomonic feature of constrictive pericarditis. The diastolic dip was present after operation in only two cases (Cases 5 and 12). In Case 5 the curve was definitely of pathologic type (Fig. 23 b). In Case 12, it was not entirely characteristic, but the shape of the dip was distinctly visible.

The *pulse pressure* in the right ventricle ranged from 10 to 42 mm Hg (Table XII), two patients having over 30 mm Hg. The ratio of end-diastolic to systolic pressure had fallen after operation to 1 : 3 or less in all except Case 5. Only two patients (Cases 16 and 36) had raised pulse pressure; their condition had improved after operation, but complete restitution had not occurred.

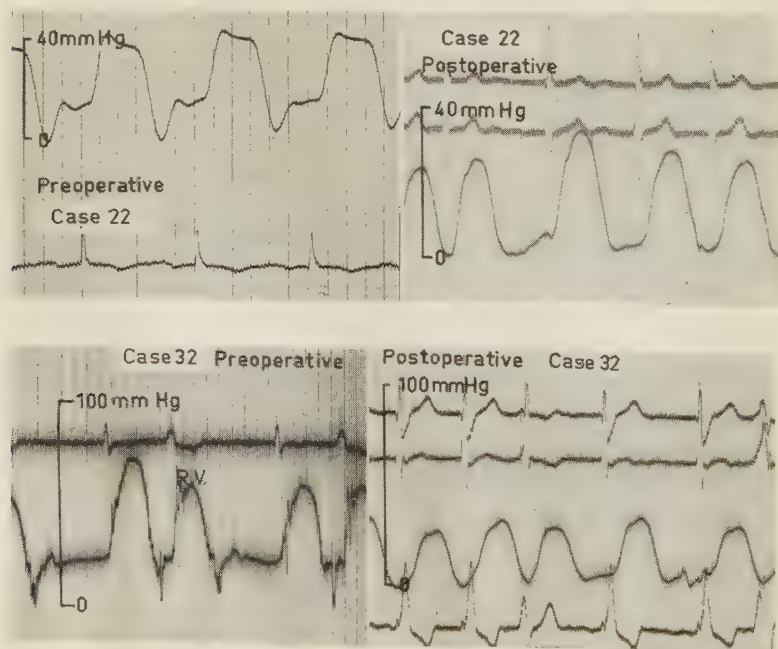


Fig. 24. Typical right ventricular pressure curves: before operation and at follow-up examination.

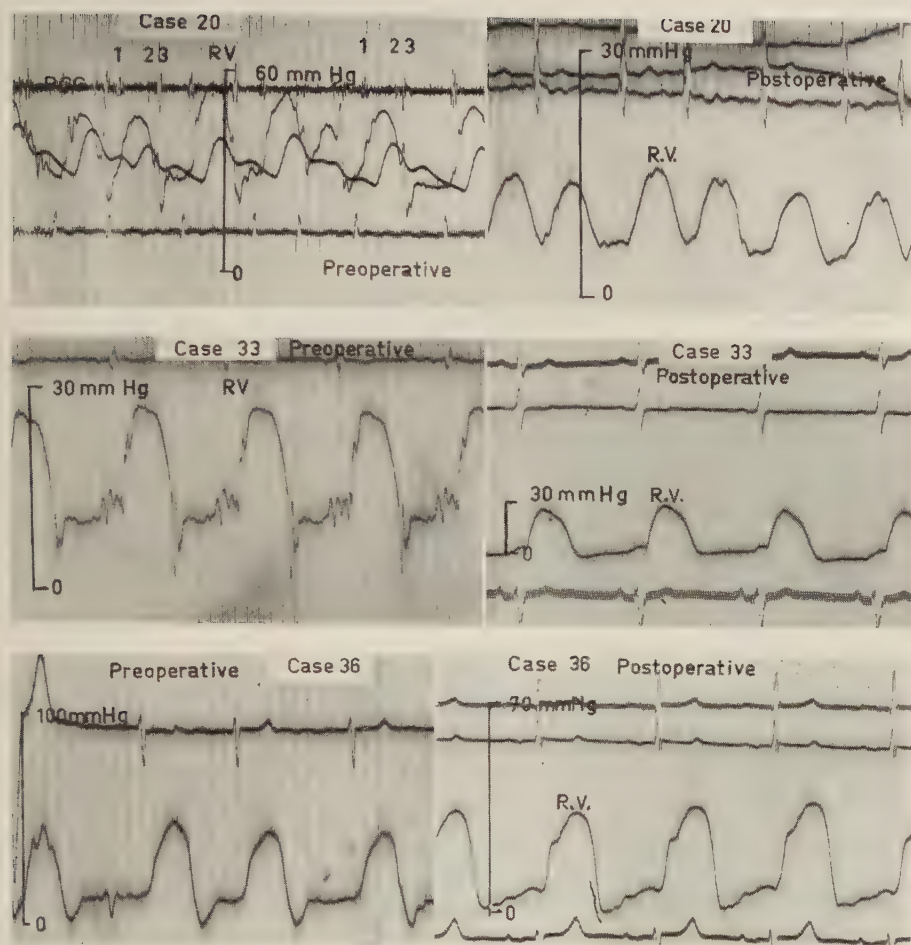


Fig. 24. (Continued.)

Thus, after successful decortication, the right ventricular curve showed a return to the normal type, with disappearance of the diastolic dip, unchanged normal pulse pressure, and a ratio of end-diastolic to systolic pressure of 1:3 or less.

Figure 24 shows some typical tracings from the right ventricle before and after decortication.

PULMONARY ARTERY PRESSURE

The mean pressure in the pulmonary artery in 27 cases was a mean 17.3 ± 1.6 mm Hg, with a range of 6 to 34 mm Hg (Table XII). The mean systolic pressure was 24.8 ± 2.1 mm Hg, ranging from 11 to 55 mm

Table XIV. Pulmonary artery pressure and pulse rate at rest and at work: pre- and postoperative values in 12 cases.

	n	Pressure mm Hg			
		Preoperative		Postoperative	
		At rest	At work	At rest	At work
P A	12	25.7 ± 1.7	36.8 ± 3.5	17.8 ± 2.9	24.5 ± 3.7
M					
Pulse/min ...		89 ± 7.7	107 ± 7.3	76 ± 4.4	94 ± 4.9

Hg. The mean diastolic pressure was 10.4 ± 1.3 mm Hg (range 3 to 28 mm Hg).

At work, the mean pulmonary artery pressure in 23 cases rose from a mean 17.1 ± 2.78 to 22.8 ± 2.33 mm Hg (Table XIII). In 12 cases, the mean pressure at rest in the pulmonary artery was 25.7 ± 1.7 mm Hg before operation, and rose at work to a mean 36.8 ± 3.5 mm Hg. Postoperatively, the mean pressure at rest in the same cases was 17.8 ± 2.9 mm Hg, rising at work to a mean 24.5 ± 3.7 mm Hg (Table XIV). In 20 patients, the pressure conditions at rest and at work lay within normal limits (Fig. 22).

PULMONARY CAPILLARY VENOUS PRESSURE

The pulmonary capillary venous pressure was recorded in 19 cases. The mean pressure was 7.6 ± 1.2 mm Hg. In one patient (Case 5) it was 25 mm Hg, the range in the remaining cases being 1 to 12 mm Hg. In 11 cases the mean PCV pressure before operation was 16.8 ± 1.14 mm Hg, the corresponding figure after operation being 5.8 ± 1.21 mm Hg (Fig. 25 and Table XII).

3. ARTERIAL OXYGEN SATURATION

In 24 cases, there was no oxygen unsaturation of the arterial blood at rest. The mean value for the oxygen saturation was 95 ± 0.44 per cent, with a range of 91 to 99 per cent.

At work, no change occurred in the mean value of the arterial oxygen saturation, *i. e.*, it was 94.2 ± 0.6 per cent, with a range of 88 to 99 per cent (22 cases).

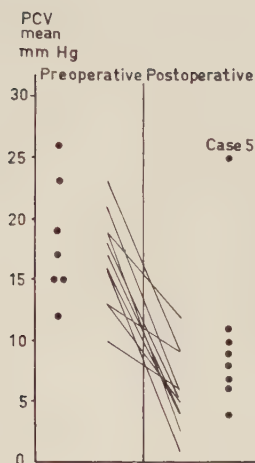


Fig. 25. PCV mean pressure before and after operation.

In the cases with the lowest values for the arterial oxygen saturation at rest before operation (Cases 12, 13 and 32), a rise was recorded postoperatively. In the remaining cases, small deviations in both directions were recorded.

4. OXYGEN UPTAKE

In 27 cases, the oxygen uptake per minute/m² of body surface ranged at rest from 84 to 291 ml.

At work, the oxygen uptake varied between 259 and 609 ml per minute/m² (24 cases). The increase lay between 66 and 430 per cent of the resting value.

The ventilation coefficient at rest was 1.39 L/100 ml to 5.68 L/100 ml, and at work ranged from 1.25 to 2.34 L/100 ml. Although the preoperative values were somewhat higher, they showed the same tendency to fall at work.

5. ARTERIOVENOUS OXYGEN DIFFERENCE

The arteriovenous oxygen difference at rest ranged from 30 to 67.3 ml/L (26 cases) with a mean value of 45.3 ± 1.9 ml/L (Table XV). It was more than 50 ml/L in 8 cases (Fig. 26).

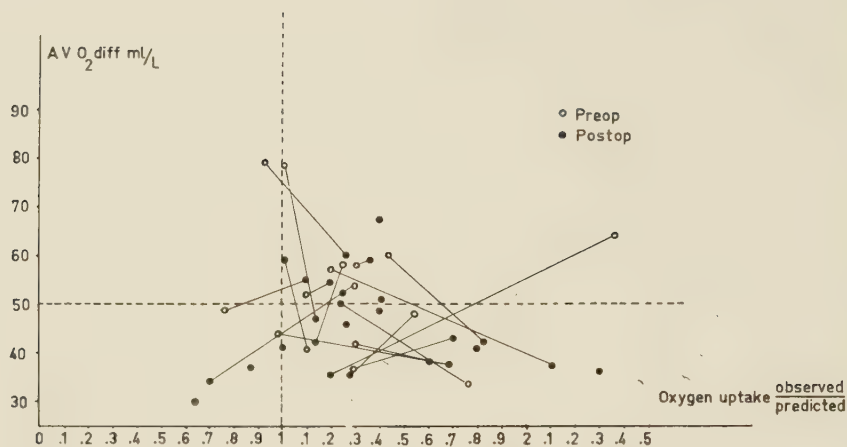


Fig. 26. Arteriovenous oxygen difference (ml/L) at rest in relation to observed/predicted oxygen uptake: before operation (circles) and after (dots). For explanation; see Figure 9.

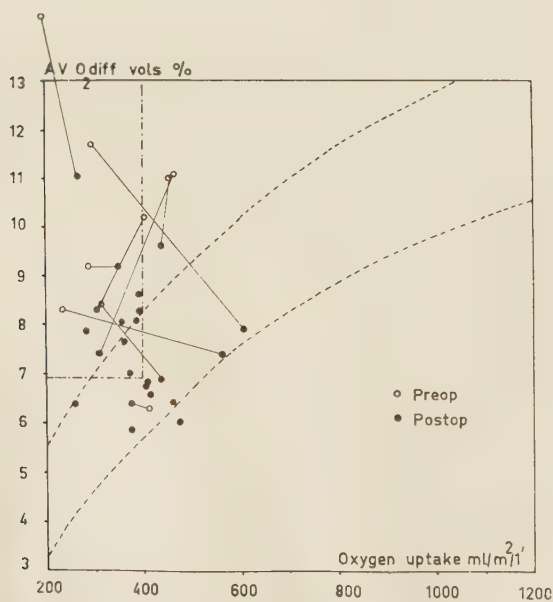


Fig. 27. Arteriovenous oxygen difference (vol. %) at work in relation to oxygen uptake; after operation. A line joins the preoperative value (circle) to the corresponding postoperative value (dot). For explanation; see Figure 10.

Table XV. Arteriovenous oxygen difference at rest and at work: pre- and postoperative values.

	n	A-V O ₂ diff ml/L At rest	n	A-V O ₂ diff ml/L At work
Preop.	24	52.7±3	18	89.6±6.1
Postop.	26	45.3±1.9	24	75.8±2.5

Table XVI. Arteriovenous oxygen difference at rest in 18 cases and at work in 11 cases: pre- and postoperative values.

	n	A-V O ₂ diff ml/L At rest	Diff p	n	A-V O ₂ diff ml/L At work	Diff p
Preop.	18	52.5±5.2	1% < p < 5%	11	96.2±7.7	1% < p < 5%
Postop.		45.5±4.4			81.7±3.9	

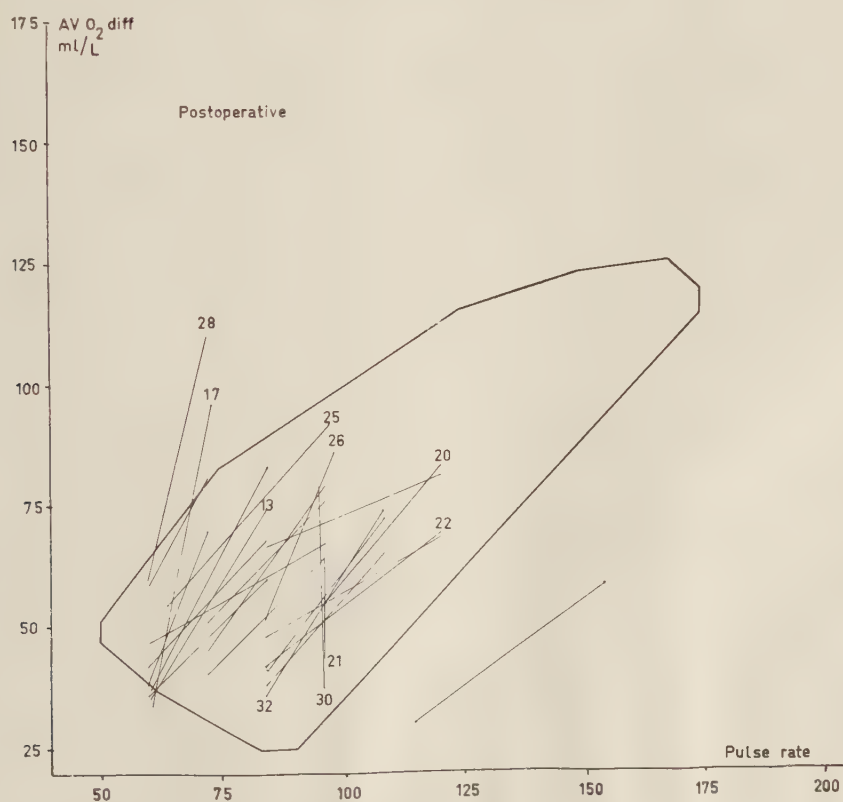


Fig. 28. Arteriovenous oxygen difference (ml/L) at rest and at work in relation to pulse rate: after operation. The numerals denote the same patients as in Figure 11. For explanation: see Figure 11.

At work, the arteriovenous oxygen difference varied between 58.5 and 110 ml/L with a mean value of 75.8 ± 2.5 ml/L in 26 cases (Table XV). The arteriovenous oxygen difference in relation to that in a series of normal cases is shown in Figure 27.

In 18 cases, the arteriovenous oxygen difference at rest was determined both before and after operation. The difference between the mean value on these two occasions is almost significant (Table XVI).

In 11 cases, the arteriovenous oxygen difference at work was recorded pre- and postoperatively. The difference between the mean value before and after operation is almost significant (Table XVI).

The relation of the arteriovenous oxygen difference to the pulse rate at rest and at work can be inferred from Fig. 28.

6. CARDIAC OUTPUT

After operation, the cardiac output in L/min/m² of body surface in relation to the oxygen uptake was within normal limits in most cases (Fig. 29).

The best criterion of successful decortication is an increase in the preoperatively low stroke volume. Postoperatively, the stroke volume at rest in 26 cases ranged from 43 to 200 ml, with a mean value of 101 ± 7.9 ml (Table XVII). In the 15 cases in which determinations were made both before and after operation, the stroke volume had increased from a mean 77 ± 10.3 ml to 110 ± 10.2 ml (Table XVIII).

At work, the stroke volume was practically unchanged, the mean value (23 cases) being 99 ± 6.7 ml, varying from 58 to 193 ml (Table XVII).

Although it is not an ideal basis of comparison, the body surface was used to reduce the effect of variations in body size in pre- and postoperative determinations of the stroke volume (Fig. 30). After operation, the stroke volume per m² of body surface (stroke index) was a mean 56 ± 4 ml at rest (26 cases), the corresponding figure at work being 56 ± 3.4 ml (23 cases): see Table XVII. In 15 cases in which determinations were made both before and after operation, the mean stroke index at rest before operation was 41.4 ± 5.1 ml; postoperatively, it was 60.3 ± 5.1 ml. This difference is significant (Table XVIII and Fig. 30).

The stroke index was also determined in relation to the pulse rate. In

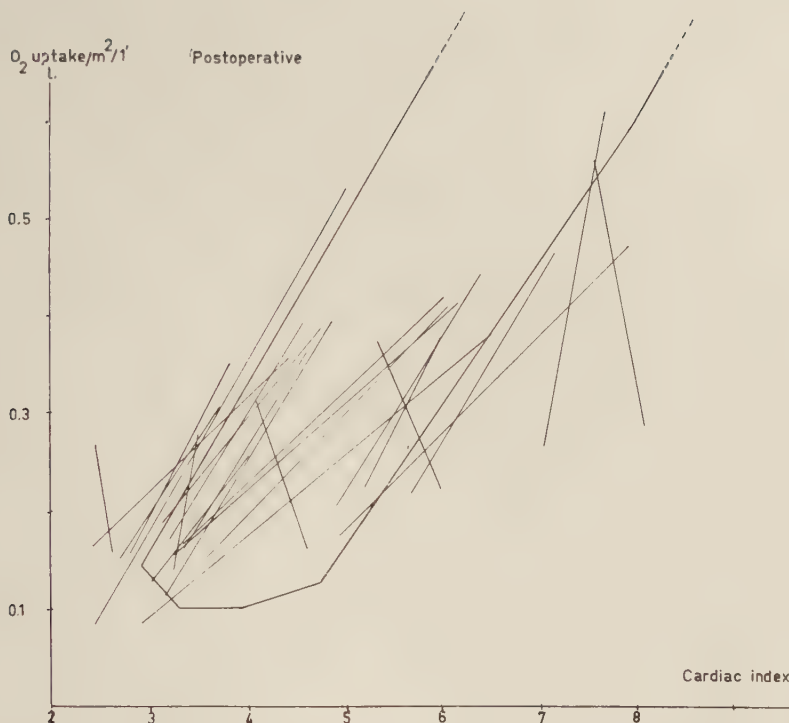


Fig. 29. Cardiac index at rest and at work in relation to oxygen uptake/min/m² of body surface: after operation. For explanation: see Figure 12.

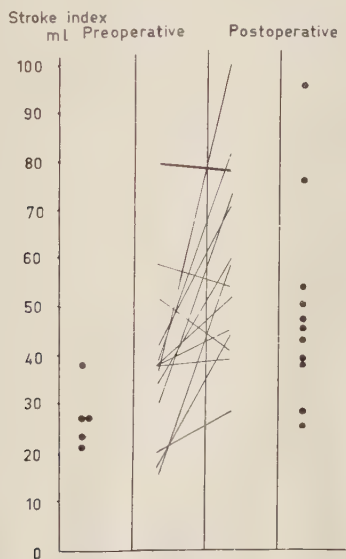


Fig. 30. Stroke index before and after operation in the whole series, and in 15 cases in which both pre- and postoperative data were available.

Table XVII. Stroke volume, stroke index and pulse rate at rest and at work: pre- and postoperative values.

	Stroke volume ml	Stroke index ml	Pulse/min
Preop.			
At rest (20 cases)	69±8.4	38±4.1	93
At work (15 cases)	59±7	33±3.5	107
Postop.			
At rest (26 cases)	101±7.9	56±4	75
At work (23 cases)	99±6.7	56±3.4	98

Table XVIII. Stroke volume, stroke index and pulse rate at rest in 15 cases: pre- and postoperative values.

	Stroke volume ml	Stroke index ml	Pulse/min
Preop.	77±10.3	41.4±5.1	88±6
Postop.	110±10.2	60.3±5.1	72±4
Diff. <i>p</i>		<i>p</i> <1 %	1 %< <i>p</i> <5 %

the 15 cases in which both pre- and postoperative data were available, the stroke index at rest was a mean 41.4 ml with a pulse rate of 88 ± 6 /minute before operation (Table XVIII and Fig. 31). After operation, the mean stroke index was 60.3 ml with a pulse rate of 72 ± 4 /minute.

In the whole series, the stroke index at rest before operation (20 cases) was 38 ml with a pulse rate of 93/minute, the corresponding figure at work (15 cases) being 33 ml with a pulse rate of 107/minute (Table XVII). Post-operatively (26 cases), the stroke index at rest was 56 ml with a pulse rate of 75/minute. At work (23 cases), which was generally twice as great as before operation, the mean stroke index amounted to 56 ml with a pulse rate of 98/minute.

Thus, in the cases in which determinations were made both before and after operation (Fig. 31), as well as in the whole series, the stroke index at rest increased after operation, with a concurrent decrease in pulse rate. This is a natural consequence of the improved filling and emptying conditions. The constricted heart reacts in a similar way to the deconstricted heart, the difference being the size of the stroke index.

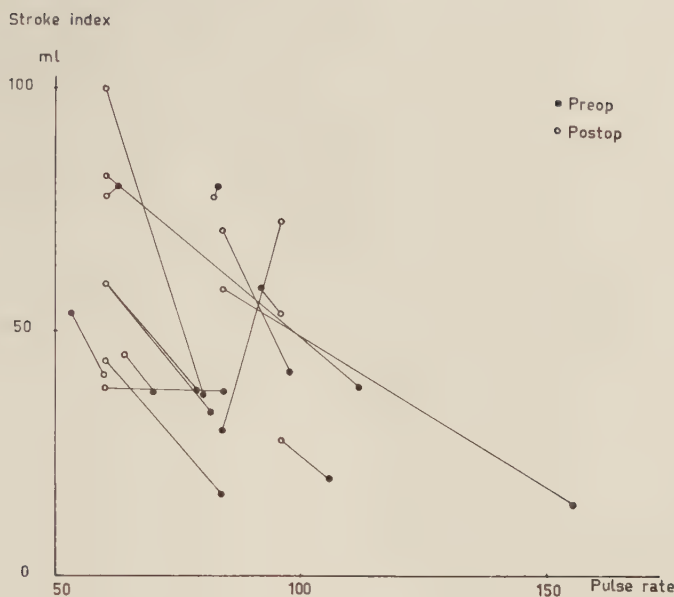


Fig. 31. Stroke index in relation to pulse rate before operation (dots) and after (circles) in 15 cases examined both pre- and postoperatively.

7. ELECTROCARDIOGRAPHY

Electrocardiograms were recorded postoperatively in 29 cases. In 9 of them, the changes observed before operation had disappeared completely, and the ECG could be denoted as normal. Improvement from the electrocardiographic point of view could be noted in some of the other 20 cases.

Postoperatively, the rhythm was irregular in 9 cases. In 8 of them, arrhythmia had been present before operation, whereas in the remaining patient (Case 22) it did not appear until after operation.

Regular sinus rhythm was present in 20 cases. In 19 of them, this also applied preoperatively; in the remaining patient (Case 36), the rhythm was regularized postoperatively by administration of quinidine sulphate.

Preoperatively, the average heart rate at rest in 28 cases was 86/min; postoperatively, the corresponding figure was 75/min.

After operation, the conduction time was completely within normal limits in 20 cases, the range being 0.12 to 0.20 sec.

Double-peaked P waves as a sign of increased atrial contraction were present in one case (Case 12). This electrocardiographic sign of a disturbance in atrial contraction had also been recorded preoperatively.

The electric axis was normally placed in 21 cases. Right axis deviation was present in 5 cases, and left axis deviation in 3. The direction of the electric axis had undergone a change in 6 cases. Thus, a normal electric axis had become deviated to the left in three cases, and to the right in two. In one case in which there was right axis deviation before operation, the axis had a normal direction after it. These changes in direction of the electric axis after operation are understandable, in view of the positional changes of the heart that may occur after disappearance of the exudate, or formation of postoperative adhesions.

Low voltage of the QRS complex was recorded in Case 5, *i. e.*, the patient in whom signs of recurrence of the constriction had appeared.

In all except one (Case 32) of the 29 patients, the QRS interval was within normal limits. In one case it had increased from 0.05 to 0.14 sec.

The T waves in the standard leads which, before operation, had been partly positive, isoelectric or negative, underwent no change after operation in 9 cases. In 11 cases, the T waves in the standard leads exhibited greater positivity.

The T waves in the chest leads showed no change in two cases. Increased positivity could be noted in 14 cases. In three of four cases in which no chest leads had been recorded preoperatively, the T waves were positive, and in one they were negative.

Changes in the S—T interval were present in four patients (Cases 11, 16, 19 and 33). One of them (Case 16), in whom such changes were present at preoperative examination, was still digitalized at the follow-up examination.

Thus, to sum up, the following statements can be made. Postoperatively, a normal ECG was recorded in 9 of 29 cases in which a pathologic ECG was recorded preoperatively. Improvement from the electrocardiographic viewpoint could be noted in additional cases.

When the follow-up examination was made, six patients were taking digitalis; thus, it did not influence the appearance of the ECG to as great an extent as before operation.

CHAPTER 16

ROENTGENOLOGIC EXAMINATION

Preoperatively, the cardiac borders were frequently masked by pleural exudate; this made roentgenologic examination more difficult, particularly with respect to evaluation of the size of the heart. Postoperatively, no exudate could be observed in either pleura in any of the 30 cases in which a roentgenologic examination was made. Obliteration of the phrenicocostal sinus on one or both sides was observed in the great majority of cases, as a sign of the former exudate and a sequel of operation. In some cases, these adhesions played a considerable role in the persisting impairment of respiratory function.

Pulmonary congestion visible roentgenologically was present in only one patient (Case 5), in whom incomplete decortication had failed to relieve the symptoms.

SIZE OF THE HEART

In six of the cases in which the heart was enlarged, the enlargement was evaluated as generalized. The left atrium was enlarged in three cases and the left ventricle in three. In one case both the left atrium and ventricle were enlarged. Enlargement of the right ventricle was present in two cases.

A considerable increase in the size of the heart occurred after operation in one patient (Case 5), who had typical symptoms and signs of constriction (Fig. 32). The heart had increased in size postoperatively in altogether seven cases. In five of them (four men and one woman) the size exceeded the normal borderline both before and after operation, whereas in one (a man) this did not occur until after operation. In one

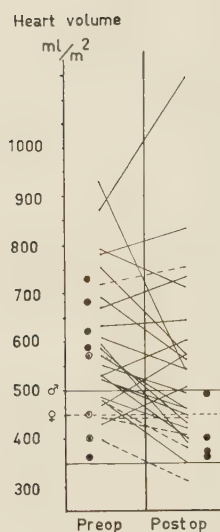


Fig. 32. Heart volume (ml/m² of body surface) before and after operation. The broken lines and ringed dots denote the women.

Table XIX. Heart volume: pre- and postoperative values (both pre- and postoperative values available in 26 cases). In the children in question, the upper normal borderline is 350 ml/m² body surface.

	n	Males ml/m ² BSA			n	Females ml/m ² BSA			
		< 500	> 500	Mean		> 350	< 450	> 450	Mean
Preop.	26	5	21	614±27	8	2	3	3	487±38
Postop.	25	14	11	539±35	5	—	4	1	457±76
Preop.	21	4	17	619±30	5	2	1	2	494±57
Postop.	21	10	11	561±39	5	—	4	1	457±76

case, also a man, the size of the heart had increased, but was within the normal range of variation both pre- and postoperatively.

In 16 cases the size of the heart decreased after operation, in three it remained unchanged, and in four cases it had not been possible to measure the cardiac borders prior to operation. The size of the heart before and after operation is shown in Table XIX and Figure 32.

PERICARDIAL CALCIFICATIONS

Postoperatively, the calcifications were seen to be reduced to the extent that freeing of the heart had been successful. Since in most cases it had not been intended to decorticate the right side of the heart, the extent of the calcifications remained unaltered on this side.

ROENTGENKYMGRAPHY AND ELECTROKYMGRAPHY

Postoperatively, the movements of the heart were studied chiefly by electrokymography, as well as at fluoroscopy in connexion with roentgenologic examination. At our hospital, roentgenkymography has not been regarded as a necessary method of examination. It has been considered that an experienced roentgenologist can equally well observe the excursions of the heart at fluoroscopy, and form an opinion on whether or not they are normal. In all the cases in which a roentgenologic examination was made, the movements of the heart were normal. In no case was there any note of exceptionally small excursions.

ELECTROKYMGRAPHY.—A postoperative examination was made in 25 cases; in 9 of them the tracings were denoted as normal or as exhibiting only inappreciable changes. A more detailed analysis could be made in 19 of the 25 cases.

RIGHT ATRIAL ELECTROKYMGRAMS

In 4 cases the right atrial electrokymogram was not studied, owing to persistent atrial fibrillation. In 2 (Cases 10 and 11) of 6 cases in which no recording had been made before operation, the tracings were normal after it. In the other 4 (Cases 3, 12, 17 and 19) signs of an impediment to emptying were present, in the form of a deepened presystolic wave with a duration of between 0.10 and 0.15 sec.

The postoperative recording showed atrial fibrillation in one patient (Case 22) with a normal tracing and cardiac rhythm before operation.

Normalization took place after operation in 5 cases. In 2 of them (Cases 27 and 33) the tracing showed no deviation from the normal. In the other 3 (Cases 21, 30 and 34), slight deviations were still present;

the duration of the presystolic segment of the curve ranged from 0.10 to 0.12 sec (preoperatively: 0.12 to 0.18 sec).

In two patients (Cases 29 and 32) with signs of an impediment to emptying on the preoperative tracing, no alteration was recorded after operation.

In one patient (Case 36) the preoperative atrial fibrillation was converted into normal sinus rhythm. The postoperative tracing then showed distinct signs of an impediment to emptying, the presystolic segment having a duration of 0.14 to 0.15 sec.

LEFT ATRIAL ELECTROKYMGRAMS

This tracing was not studied in 4 cases of atrial fibrillation.

No preoperative recording had been made in 6 patients. In 3 of them (Cases 3, 11 and 27) the curves were normal after operation. In the other 3 (Cases 10, 12 and 17) there were signs of an impediment to emptying of varying severity, with a duration of the deep presystolic part of the curve ranging from 0.09 to 0.12 sec.

Three patients (Cases 29, 31 and 34) had a normal electrokymogram before operation, and no change was recorded after it.

An impediment to emptying was present preoperatively in 5 cases. In 3 of them (Cases 21, 22 and 33) the postoperative tracings could not be analyzed for various reasons, such as fibrillation, a high pulse rate or incomplete recording. In the other 2 (Cases 28 and 30) the signs of impeded emptying persisted. In one of them the duration of the presystolic deflexion was unchanged and amounted to 0.16 sec. In the other, signs of less emptying difficulty were apparent, the duration of the presystolic deflexion having decreased from between 0.10 and 0.12 sec to between 0.07 and 0.10 sec.

In one case (Case 36) in which fibrillation had become regularized after operation, a pathologic curve was recorded, the duration of the presystolic deflexion being 0.12 to 0.14 sec.

RIGHT VENTRICULAR ELECTROKYMGRAMS

In 8 cases the right ventricular electrokymogram was not recorded before operation, or the tracings were unsatisfactory. Postoperatively, the filling phase was more rapid than the emptying phase in 6 of these patients (Cases 3, 12, 16, 17, 20 and 26), whereas the tracing was normal

in the other 2 (Cases 10 and 11). Two patients (Cases 33 and 34) had a normal right ventricular electrokymogram both pre- and postoperatively.

Definitely or faintly pathologic features were present preoperatively in 9 cases. They persisted after operation in one patient (Case 22), disappeared in 7 (Cases 21, 27—31 and 36), and in one patient (Case 32) no recording was made postoperatively.

LEFT VENTRICULAR ELECTROKYMOGRAMS

The left ventricular electrokymogram was not recorded before operation or was unsatisfactory in 6 cases. In 5 of these patients (Cases 3, 10, 12, 16 and 17) the postoperative tracing was pathologic to a varying degree, and in the remaining case (Case 11) it was normal.

Abnormal pulsations of the left ventricle were present before operation in 12 cases. In 5 of them (Cases 20, 22, 27, 31 and 36) the pathologic features persisted more or less distinctly after operation, whereas in 7 (Cases 21, 27—30, 32 and 34) the tracing was normal. One patient (Case 33) had normal pulsations both pre- and postoperatively.

COMMENTS

In view of the marked clinical improvement in most cases, it is surprising that signs of disturbances in atrial or ventricular action were present in so many patients after operation. Completely normal tracings in all segments were recorded in only three cases (Cases 11, 27 and 33); two of them had pathologic curves before operation, and in Case 11 no electrokymographic examination had been made preoperatively. On the other hand, the pathologic features had decreased to a great extent in some cases (Cases 21, 28—31 and 34).

A tendency to normalization was found more often in the ventricular than in the atrial tracings. This could be expected in view of the fact that the object of operation was to decorticate the ventricular region. It is therefore of some interest to note that, although the right atrium was not decorticated, normal electrokymograms could be recorded after operation, as a result of normalization of the circulation (*e. g.* Cases 27 and 33). But, on the other hand, the pathologic changes in the right atrial electrokymogram sometimes persisted despite normal circulatory conditions after operation. This can certainly be ascribed partly to myocardial fibrosis. As far as the atrial tracings were concerned, normal

circulation was evidently not necessarily equivalent to normalization of the electrokymogram, whereas in the ventricle these two events almost invariably ran parallel.

ANGIOCARDIOGRAPHY

Postoperative angiocardiology could be performed in only two patients (Cases 11 and 27). In Case 11 the passage through the heart was more rapid than before operation, and the preoperatively normal anatomic conditions in the heart and great vessels had not undergone any change. In Case 27 angiocardiology was performed to ascertain whether any local impediment was responsible for the persistent liver enlargement, but no such changes could be observed.

CHAPTER 17

GENERAL DISCUSSION

SOME PHYSIOLOGIC ASPECTS AND DIAGNOSIS

In most cases, constrictive pericarditis presents no diagnostic difficulties. As BLOUNT *et al.* (cit. BURWELL) and BURWELL *et al.* have pointed out, it is only fibrosis of the myocardium that may simulate constrictive pericarditis exactly, and that cannot be distinguished from it by cardiac catheterization. Consequently, there is sometimes no other course than to explore the pericardium, to avoid overlooking the presence of constrictive pericarditis, and depriving the patient of the possibility of operation.

Both clinical and experimental studies have shown that even if some effect on systolic emptying cannot be ruled out, it is chiefly limitation of diastolic filling, with resulting low stroke volume, that is responsible for the disturbance in heart function. This impeded filling leads to an increase in filling pressure in both ventricles, and is reflected in the systemic circulation as high venous pressure, and in the pulmonary circulation as raised PCV pressure. The rise in filling pressure in both sides of the heart indicates that the locally constricting scar tissue does not play as great a role as has been believed.

The high filling pressure in the left ventricle, and dilatation of the left atrium, together with the electrokymographic signs of an impediment to emptying of the atrium, explain why constrictive pericarditis resembles mitral stenosis. Naturally, this apparent resemblance is not due to changes in the atrioventricular region on the left side — except in extremely rare cases — but is to be ascribed to the impediment to diastolic filling of the left ventricle, as a result of the pericardial lesions.

The arteriovenous oxygen difference is high both at rest and at work, and the low stroke volume leads to a low cardiac output. Individual variations in the degree of effect of the pericardial changes are illustrated by Cases 24, 28, 30, 31 and 35 on the one hand, and Cases 20, 21, 27 and 29 on the other. In the former cases, the cardiac output could not be increased at work. In Cases 23 and 24, considerable tachycardia was present at rest; it did not increase at work, and the cardiac output therefore remained unaltered.

Diagnostic signs of constrictive pericarditis include the well-known symptom complex of venous stasis with cyanosis, oedema, ascites and exertional dyspnoea. The pulse pressure is low, and a paradoxical pulse can sometimes be observed. The heart, which is the least conspicuous feature, may have a third sound — protodiastolic gallop rhythm — and a murmur is usually lacking. The presence of any marked murmur is generally indicative of some form of valvular disease. Typical although not pathognomonic pressure curves at cardiac catheterization may afford further evidence. A low serum protein level, inappreciable disturbances in liver function, low voltage and changes in the T waves on the electrocardiogram, typical electrokymograms and the presence of pericardial calcifications complete the diagnosis.

SOME CONSIDERATIONS ON SURGICAL TREATMENT

The patients are generally handicapped by the disease; in the present series all the patients were, in fact, hospital cases with impairment of physical working capacity. Spontaneous improvement seldom occurs, and WARBURG has stated that once the symptoms appear, the expectation of life usually does not exceed 10 years. If, on the other hand, operation is successful, there are good prospects of the patient being able to resume a normal, active life. The great difficulty lies in selecting suitable cases for operation.

The immediate outcome of operation and the future outlook are determined by the functional state of the myocardium. No better functional test exists than the ability of the myocardium to tolerate the operation, but this test is obviously inapplicable for evaluating the risks of operation before it takes place.

The appearance of the ventricular pressure curve has been stated to be a criterion of myocardial function. A diastolic dip to zero level has

been regarded as an expression of satisfactory function. Seen against this background, two of the postoperative deaths in the present series (Cases 18 and 23), with pressure curves of the type indicative of a poor state of the myocardium, would be explainable. But the conditions of size, contraction and emptying of a ventricle surrounded by a pathologic pericardium are unquestionably a complex matter. Let us assume that an angiocardiographically enlarged ventricle, with a large quantity of residual blood at the end of systole, is a sign of a poorly functioning myocardium, and that the constricting pericardium mainly affects diastolic filling. In this event, Cases 35 and 36, for example, had a poor myocardium from the angiocardiographic point of view, but an efficient myocardium judging by the type of ventricular pressure curve. Moreover, since in these cases operation disclosed severe myocardial lesions, and deeply penetrating calcifications which could not be removed, there was additional evidence of chronic myocarditis, despite the type of ventricular pressure curve. The outcome of operation nevertheless indicated that myocardial function was satisfactory.

A study of the operative deaths also fails to provide any definite contraindications to operation on the basis of the preoperative prognosis. The tendency in recent years has been to base the evaluation on the conditions of size and emptying in both sides of the heart. Only unsatisfactory function of the left ventricle, as seen on the angiocardiograms, has been regarded as a stronger contraindication to operation.

A prerequisite for satisfactory results of operation is a proper understanding of the influence of the pericardium on circulation. The haemodynamic conditions in constrictive pericarditis indicate that the primary necessity is to decorticate the ventricular region. Decortication from the right side alone must be regarded as incorrect, in view of the fact that only a limited portion of the pericardium, of little pathophysiologic importance, is accessible with this approach (Fig. 33). This is apt to result in anatomically inadequate decortication, with persisting constriction (*e. g.* Case 5), even if skilful surgeons have succeeded in freeing the heart with this approach (*e. g.* Case 1). Decortication of the right side at a later session was carried out in only one case in the present series (Case 12) and, judging by the pressure conditions, was not fully warranted.

The superiority of left-sided thoracotomy to the sternum-splitting incision has already been emphasized. But the stability of the thorax after operation is not the only advantage. The difference between the two

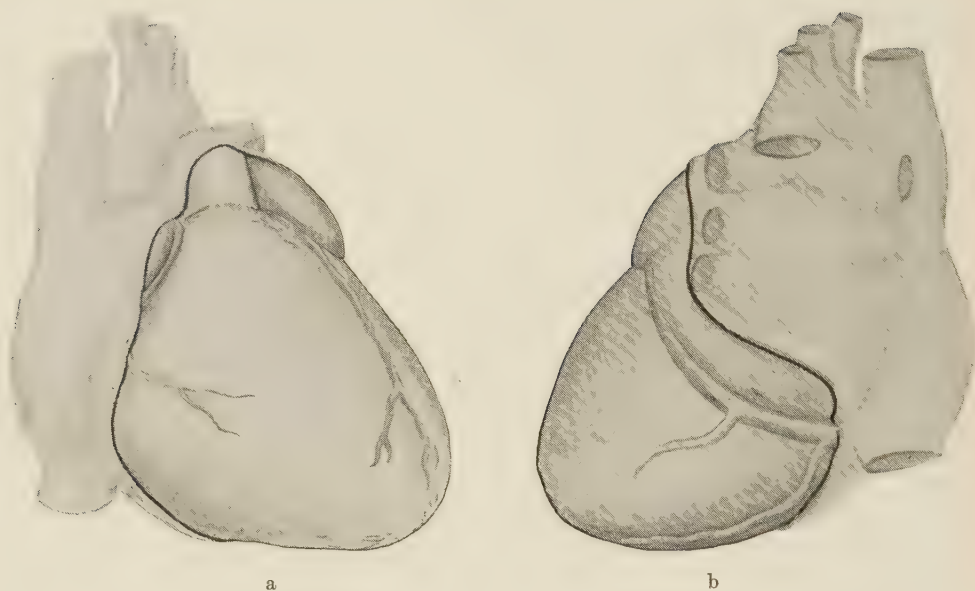


Fig. 33. The extent to which pericardial resection is possible through the left transpleural approach.
 a. anterior aspect of the heart.
 b. posterior aspect of the heart.

techniques is most apparent at operation. In order to decorticate the region of the heart behind the easily accessible parts (beyond the borders) it is necessary, when using an anterior incision, to lift the heart upwards. This produces kinking of the large vessels at the base, with a resulting impairment of circulation and fall in blood pressure. This contributes to increasing the arrhythmias that are always easily elicited from the apex region during decortication.

In this respect, left-sided decortication is far more lenient, since it is seldom necessary to displace the heart to such an extent for its anterior or posterior aspect to be accessible. Actually, the question of decortication beyond the borders scarcely arises, since the whole operative field is easily surveyed and accessible. After decortication, a piece of pericardium remains in both cases; with the sternum-splitting incision it is attached to the posterior aspect of the heart, and in left-sided decortication it covers the venae cavae and the right atrium (Fig. 33). From the functional point of view, it seems preferable for the portion to be left *in situ* to be on the right side, where it has least influence on the contractility of the heart.

It is, however, only under ideal conditions that such extensive decortication can be performed, since the pericardial changes, with calcifications extending to a varying depth into the myocardium, make it necessary to compromise. Operation consists of removal not only of the outer layer of pericardium, but also of the visceral portion, which may be involved and thus form an impediment to diastolic filling. This portion, which possibly consists of an organized layer of exudate adherent to the visceral pericardium, may be so firmly attached to the myocardium that its removal is a technical impossibility.

The type of operation and the extent to which it has been successful in freeing the ventricle from the adherent pericardium are among the most important external factors for potential restitution of function. No comparison can be made between the results of the different types of operation in the present series, since left-sided decortication was performed in the majority of cases.

RESULTS

BURWELL, who has a long experience of constrictive pericarditis, has stated that most patients are improved after operation, and many resume normal activity. He nevertheless pointed out that, if periodic examinations are made, it is found that normal circulatory conditions have seldom been restored. Even if the patients can live a normal life, the haemodynamic conditions are still pathologic.

These statements might seem to be inapplicable to the present series, in which normal pressure conditions and a marked rise in cardiac output were recorded in more than half of the patients. It is, however, found that the more numerous the investigations made, the fewer patients remain who can be said to have normal circulatory conditions. Less thorough investigation must imply that those who have considered themselves able to work are classified as recovered, whereas a thorough investigation, using all the means available for circulatory studies, sifts out all those who have only more or less recovered. It may therefore appear questionable to report the results of an investigation in which the expectations were not fulfilled.

It cannot, however, be denied that many patients derived considerable benefit from operation. Thus, 26 of them were able to resume a normal occupation, and only one of these patients is still under control for circulatory complaints. One patient worked as a lumberman, two had

engaged in strenuous sports, and the remainder had demands on their physical working capacity corresponding to those of most people. Some of these patients had myocardial changes of such a nature that not even complete decortication would have been able to restore normal conditions. None of them had signs of persisting constriction.

The improvement in circulation time, stroke volume and pressure was the most striking feature. But if all the circulatory data, such as cardiac output at rest and on slight exertion, arteriovenous oxygen difference and pressure under corresponding conditions are assembled, it is found that at least 12 patients had normal circulation, and probably another three as well, in whom complete data were lacking. An improvement had also taken place in the other cases, but some of the functions were still within pathologic limits.

Four patients were unable to resume work (Cases 12, 26, 27 and 35). This was due in the respective cases to myocardial disease and constricting pleural lesions; fibrosis of the myocardium; cirrhosis of the liver and respiratory insufficiency; and sequelae of a cerebral haemorrhage.

One patient (Case 5) had typical symptoms and signs of constrictive pericarditis at follow-up examination.

The electrocardiographic changes had regressed in some cases, but persisted in the majority. After exclusion of four cases with persisting ECG changes, 8 of the 12 patients with normal circulatory conditions were found to have a normal ECG as well. In four of these 8 cases, the result of one or another laboratory test was on the borderline of pathologic values. Thus, two patients had a raised thymol turbidity value, which could scarcely have had any association with the primary disease. In one of them, it was almost certainly due to fatty degeneration of the liver on an alcoholic basis. In the other, it was due to biliary tract disease, for which operation was later performed. The liver was then found to have a normal macroscopic appearance, and the thymol turbidity subsequently returned to a normal value. These two patients have therefore been included among the recoveries.

Tests of respiratory function showed it to be normal in one of these 8 patients, and three had values lying only slightly below normal.

Roentgenologic examination showed normal conditions in three of these four patients.

These three patients were also completely healthy from the clinical point of view. In an additional three cases, it is highly probable that the results of functional tests were normal, but since the investigation was incomplete, definite evidence is lacking.

On the basis merely of determination of the circulation time, only three of the 26 patients who had resumed work would have been classified as not recovered. On the basis of circulatory studies, 12 would have been healthy, whereas only three (possibly six) actually exhibited neither clinical nor physiologic signs of disease. Consequently, it can be agreed with BURWELL that the results of operation need to be evaluated and analyzed.

It must, however, be borne in mind that the importance to be ascribed to the various tests varies considerably. Moreover, the tests cannot always be expected to show normal values, despite improvement. Circulatory studies by means of catheterization must unquestionably be regarded as being of prime importance, and the normal or pathologic conditions then recorded cannot be disregarded. Electrocardiography and tests of respiratory function, for example, must be seen in a somewhat different light. Electrocardiographic signs of myocardial damage can scarcely be expected to disappear after pericardectomy, nor can pre- or postoperative pleural lesions fail to be reflected in tests of respiratory function.

FACTORS INFLUENCING THE END-RESULTS

If the type of operation and extent of decortication are among the most important external factors contributing to favourable results of operation, the subsequent course is influenced by several internal factors, some of which are a result of constrictive pericarditis, whereas others have no direct relation to it. As stated earlier, even a perfect operation cannot lead to improvement if the myocardium is the site of diffuse fibrosis. This diagnosis cannot be made prior to operation, since no existing method of examination can disclose the presence of this condition.

Pleural thickening is another form of fibrosis which plays a considerable role not only for the immediate results, but also for the later course. Pleural thickening was present in about 80 per cent of the cases in which a postoperative examination was made, but was of any consequence only in some of them. The postoperative exudate may give rise to thickening on the inner surface of the thorax and in the phrenicocostal sinus. This, combined with remains of the preoperative lesions, may bring about a considerable decrease in respiratory function. One of the operative deaths was, in fact, due to underestimation of the effect of this thickening on respiratory function. In at least two of the patients, poor respiratory function — combined in one case with a marked rise in the residual

quotient — was responsible for appreciable functional impairment after operation. In most cases an improvement in respiratory function could be noted in comparison to the preoperative values, but in some cases a deterioration took place.

Fibrosis of the liver and its vessels, particularly in cases with marked, long-standing stasis, has also been regarded as a factor influencing the general condition after removal of the constriction. Persisting symptoms which might have been due to a disturbance in liver function were observed in only one of the present cases. As a rule, liver function was only inappreciably impaired, despite considerable enlargement of the liver on palpation, and had no tendency to affect the general health.

Atrial fibrillation is apt to appear in constrictive pericarditis, both before and after operation. Since the pressure conditions showed less tendency to normalization in these patients than in the others, the arrhythmia probably had some effect on the subsequent course.

Pericardial calcifications were present in almost five-eighths of the present cases, but were not regarded as a contraindication to operation. However, since they often involved the myocardium as well, they increased the risks of operation and made it more difficult from the technical point of view. Although calcifications left in the myocardium are not of particularly great functional importance, localization of the underlying process to the myocardium may be a factor of importance for the future.

Bacterial infection is the most common cause of constrictive pericarditis. The presence or absence of continued activity of the causative organisms may have some influence on the future course. This is highly dependent on whether suitable bacteriostatic agents are available against infection in general and tuberculosis in particular. In the present series, active pericardial tuberculosis responded to adequate therapy, but was fatal when such therapy could not be given.

Other diseases unrelated to constrictive pericarditis may affect the condition after operation. In the present series, such diseases were emphysema and, possibly, valvular disease.

No patient was refused operation on account of advanced age, if constrictive pericarditis had produced appreciable functional impairment. Owing to coincident factors, such as changes due to the aging process and fibrosis of the myocardium, the persisting impairment of function increases with rising age.

It is essential, for proper management of the patient in the years following operation, to bear all these complicating factors in mind. This

is especially because, as has been pointed out by BURWELL, operation is not a curative measure, but is only palliative. Surgical treatment, performed as described here, nevertheless produced improvement in a not inconsiderable number of patients, even if they exhibited slight, persisting anomalies incompatible with normal circulation. Left-sided decortication has thus proved to be adequate, and none of the patients had any symptoms or signs of constrictive pericarditis after it.

GENERAL SUMMARY

CHAPTER 1. — It can be inferred from the clinical and experimental evidence reviewed that the main effect of constrictive pericarditis is to hamper ventricular function, particularly diastolic expansion. Local obstruction is seldom the cause of circulatory disturbances. Cardiac catheterization is the method of choice for determining both the presence of local constriction and the extent of the constrictive process. This information is decisive for the extent of decortication.

CHAPTER 2. — An account is given of the aetiologic factors in the 38 cases of chronic constrictive pericarditis in this series. Tuberculosis is found to be the most common cause. The aetiologic factors in all 38 cases are presented in figure 1.

CHAPTERS 4 and 13. — The clinical features before and after operation are described. The characteristic symptoms and signs are: venous stasis, cyanosis, oedema, dyspnoea, enlargement of the liver, ascites, protodiastolic triple heart rhythm, and thoracic wall phenomena. After operation, these symptoms and signs are found to have disappeared in almost every case. At follow-up examination, the constrictive syndrome was present in only one patient, in whom right-sided decortication had been performed.

CHAPTERS 5 and 14. — After operation, the most striking changes in the laboratory tests are found to be a rise in the previously low serum protein level, and an almost significant rise in the serum albumin fraction.

Postoperatively, an increase in vital capacity could generally be noted, although in a few cases it had decreased, due to pleural thickening and obliteration of the phrenicocostal space.

CHAPTERS 6 and 15. — An account is given of the haemodynamic studies. In comparable cases, the following changes are found to have taken place after operation:

The circulation time fell from 23 ± 2 to 14 ± 1 sec.

The mean pressure decreased as follows:

Right atrium: from 15.6 ± 1.7 to 4.8 ± 0.3 mm Hg

Pulmonary artery: from 25.7 ± 1.7 to 17.8 ± 2.9 mm Hg

PCV: from 16.8 ± 1.1 to 5.8 ± 1.2 mm Hg

The stroke index increased from 41.4 ± 5.1 ml to 60.3 ± 5.1 ml.

The typical changes in shape of the atrial pressure curve persisted to some extent after operation, whereas the diastolic dip and plateau in the right ventricular curve had almost invariably disappeared.

Normalization of the electrocardiogram took place after operation in nine cases, and in the remainder an improvement could be noted.

CHAPTERS 7 and 16. — The results of roentgenologic examination are described. Pleural exudate was a common finding before operation, and disappeared after it. The heart was generally larger than normal, and decreased in size after operation. Pericardial calcifications were present in 22 of the 38 cases. The typical electrokymographic changes disappeared only to some extent postoperatively. *The most important observation is that angiocardiography disclosed no stenotic processes, either intracardiac or in the great vessels.*

CHAPTER 10. — The surgical technique is described. The value of the left transpleural approach is stressed. Its main advantages are pointed out, *i. e.*, stability of the chest wall, good prerequisites for healing, and a good approach to all parts of the heart to be decorticated, as well as less risk of producing cardiac arrhythmias. Decortication through the left transpleural approach, originally intended as the first of two sessions, has proved to be adequate in every case. Right-sided decortication at a second session has not been necessary. No signs of persisting constriction were observed in any case of left-sided decortication.

The most common observation at follow-up examination is found to be a normalization of the pressure conditions, which took place in more than half of the cases. This indicates that normal circulatory conditions have been restored to a great extent, even if certain changes incompatible with normal haemodynamic findings may persist.

TABULAR SURVEY OF
CLINICAL, LABORATORY, CIRCULATORY AND
ROENTGENOLOGIC DATA

PREOPERATIVE

Case No.	Sex	Age	Body Surface Area m ²	Heart volume ml m ² BSA	Circulation time sec	Arterial O ₂ saturation %	O ₂ uptake ml min m ² BSA	AV O ₂ diff. ml/L	Cardiac output L/min	Pulse rate	Stroke index ml
1	M	42	1.90	470	17						
2	M	41	1.90	680							
3	F	11	1.10	396	7						
4	F	17	1.40	440	25						
5	M	50	1.90	870	15						
6	M	48	1.73	550	24						
7 r	M	31	1.85	790			135	78	3.2		
8	M	51	2.0	360	35						
9	M	26	1.89		25						
10 r	M	32	1.80	588	12	95.5	174	42	7.5	98	42
11	M	45	1.88	430	18						
12 r	M	29	1.66	930	15	88.5	172	58	4.94	79	38
13 r w	M	27	2.0	610	30	88.4 93.4	324 465	64 111	10.1 8.45	63 82	80 52
14	M	9	0.81								
15	M	32	2.0	520	20						
16	F	45	1.63	716	38						
17 r w	M	49	1.80	570	27		157 457	54 110	5.2 7.5	53 75	54 56
18 r w	M	20	1.90	730	28		145 258	61 96	4.5 5.1	104 135	23 20
19	M	27									

r = at rest
w = at work

POSTOPERATIVE

Case No.	Body Surface Area m ²	Heart volume ml	Circulation time sec	Arterial O ₂ saturation %	O ₂ uptake ml min m ² BSA	AV O ₂ diff. ml/L	Cardiac output L/min	Pulse rate	Stroke index ml
		m ² BSA							
1 r	1.70	570	13	94.0	187	48.5	6.55	84	39
w				94.8	419	68.2	10.2	120	50
2 †									
3 r	1.60	310	10		127	41.0	4.95	72	43
w					259	63.5	6.54	96	43
4 r	1.70	405	14		260			72	
w					447			108	
5 r	1.90	1,140	25	96.0	161	67.3	4.53	84	28
w				90.1	358	80.9	8.4	120	37
6 r	1.70	490	12	96.8	146	45.1	5.52	72	45
w				96.3	278	79.4	5.96	96	36
7 r	1.75	710	15	97.5	152	47.0	5.66	60	54
w				92.4	413	66.5	10.8	96	64
8 †									
9 r	1.84	490	13					84	
w								96	
10 r	1.86	430	12	94.0	215	38.1	10.5	84	71
w				94.5	461	64.6	13.3	108	66
11 r	1.92	500	14	94.0	111	37.0	5.78	60	50
w				98.0	395	83.0	9.14	84	57
12 r	1.66	540	12	93.3	184	59.0	5.17	60	52
w				91.0	385	81.0	7.89	72	66
13 r	1.98	540	12	96.5	163	35.4	9.12	60	78
w				95.5	310	74.3	8.26	84	49
14	1.11	370	12.7						
15 r	2.17	460	6		257	40.6	13.74	84	76
w						72.1		108	
16 r	1.63	750	19	98.8	171	51.0	5.45	72	47
w				94.2	364	76.5	7.8	96	50
17 r	1.74	470	14	94.3	83	34.0	4.25	60	41
w				92.6	440	96.0	8.0	72	61
18 †									
19 r	1.70	400	15	98.4	87	30.0	4.93	115	25
w				97.3	383	58.5	11.1	154	43

r = at rest
w = at work

(PREOPERATIVE)

Case No.	Sex	Age	Body Surface Area m ²	Heart volyme ml m ² BSA	Circulation time sec	Arterial O ₂ saturation %	O ₂ uptake ml min m ² BSA	AV O ₂ diff. ml/L	Cardiac output L/min	Pulse rate	Stroke index ml
20	r w	M	31	1.87	750	91.6 95.1	105 405	49 102	4.0 7.42	106 119	20 33
21	r w	M	25	1.78	590	98.0 100.0	170 413	35.2 62.7	8.6 11.7	92 115	59 57
22	r w	F	63	1.64	440	98.2 99.4	145 313	60 84	3.96 6.12	156 144	15 26
23	r w	F	51	1.50	570	88.8 89.8	160 318	48.5 83	4.94 5.73	156 120	21 32
24	r w	F	38	1.38	450	93.2 92.5	138 217	38 61	5.35 4.92	146 155	27 23
25	r w	M	45	1.74	630	94.4 92.0	137 287	52 92	4.6 5.43	70 84	38 37
26	r w	M	39	1.83	530	94.8 91.1		48.1 101		110 150	
27	r w	M	40	1.94	485	94.3	223 400	33.6 48.6	12.9 16.0	83	80
28	r w	M	58	1.80		92.8 96.4	113 196	79 143	2.57 2.55	84 96	17 15
29	r	M	37	1.82	530	95.5	161	58.1	5.04	82	34
30	r w	M	54	1.66	480		143 298	57 117	4.16 4.22	84 96	30 27
31	r w	F	29	1.91	480	99.6 100.0	114 218	36 64	6.03 6.5	84 95	38 36
32	r w	M	49	1.60	780	90.8 90.8	235	43 83	4.53	60 108	26
33	r w	M	34	2.05	690	97.4 99.8	129	44 53	6.03	80 84	37
34	r	M	27	1.96	540	94.0	210	48.3	8.5	112	39
35	r w	M	56	1.72	580	97.5 97.4	121 185	85 135	2.44 2.36	52 84	27 16
36	r w	M	48	2.0	670	94.5 93.6	129 341	41 67	6.3 10.2	84 120	38 43
37	r	M	31	1.45	620	94.0		56		120	
38		F	45	1.71	400						

r = at rest
w = at work

(POSTOPERATIVE)

Case No.	Body Surface Area m ²	Heart volyme ml m ² BSA	Circulation time sec	Arterial O ₂ saturation %	O ₂ uptake ml min m ² BSA	AV O ₂ diff. ml/L	Cardiac output L/min	Pulse rate	Stroke index ml
20 r w	1.87	570		92.4 92.1	149 307	55.0 83.0	5.03 6.92	96 120	28 31
21 r w	1.67	390	14	97.0 88.0	225 380	43.0 64.0	8.7 10.0	96 96	54 62
22 r w	1.64	440		91.0 94.0	205 443	42.0 69.0	8.02 10.5	84 120	59 54
23 †									
24 †									
25 r w	1.74	640		96.8 89.7	156 351	54.7 92.1	4.97 6.64	64 97	45 39
26 r w	1.82	600	14	92.0 95.0	167 392	52.3 86.0	5.80 8.30	84 98	38 47
27 r	1.94	420	10	93.5	162	50.0	6.4	82	78
28 r w	1.86	360	15	93.0 91.8	158 269	60.0 110.0	4.88 4.54	60 72	44 34
29 r w	1.77	400	12	96.9 97.3	151 410	42.0 68.0	6.37 10.7	60 84	60 72
30 r w	1.61	350	13	95.0 99.0	264 609	37.5 79.3	11.35 12.36	96 96	73 80
31	1.79	380							
32 r w	1.64	830	20	97.5 96.0	292 565	36.0 74.1	13.28 12.47	84 108	96 70
33 r w	2.0	560	13	93.5 95.8	226 375	37.8 70.3	12.0 10.7	60 72	100 75
34 r w	2.05	450	13	95.0 96.0	177 476	35.8 60.1	10.12 16.22	60 84	82 94
35									
36 r	2.0	730	20	93.0	137	58.2	4.69	60	39
37 †									
38 †									

r = at rest
w = at work

PREOPERATIVE

Case No.	Blood Pressure mm Hg								Work load Kgm/min
	Venous	R A M	R V		P A			PCV M	
			Syst.	End-diast.	Syst.	M	Diast.		
1	20								
2	22								
3	21.3								
4	20								
5	14.3								
6	27.2								
7		15.4		25 (M)					
8	18.4								
9	26								
10		23		26 (M)		31			
11		11		15 (M)		18			
12		21	32	24	30	23	20		
13 r w		24		25 (M)		31 39			
14	15								
15 r w		13		19 (M)		21 26			90
16 r w		37	64	28	23 76	19 68	14 58	19	
17 r w		7	23	14	30 55	21 41	16 29	17	75
18 r w		20	35	21	36 45	26 37	21 29		75
19									
20 r w		17	32	17	34 42	27 35	20 27	23	75

r = at rest

w = at work

POSTOPERATIVE

Case No.	Blood Pressure mm Hg								Work load Kg/min	
	Venous	R A M	R V		P A			PCV M		
			Syst.	End-diast.	Syst.	M	Diast.			
1 r w	10	—2	19	—0.4	11 24	6 18	3 11	7 10	75	
2										
3 r w		0	22	1	17 27	10 13	4 5	4	150	
4 r w		9	31	8	29 29	21 21	13 14	10	125	
5 r w		17	39	16	46 57	33 45	25 35	25	75	
6 r w		7	23	5	27 33	20 24	13 18	11	150	
7 r w		10	36	11	34 37	24 27	16 17		150	
8 r										
9 r w			4 6	24	9					75
10 r w			9	31	11	24 32	17 24	9 14		150
11 r	7					15			150	
12 r w		7			18 27	13 17	8 11	9	75	
13 r w		5	16	3	19 21	11 10	5 5	6	75	
14 r										
15 r w		4	16	5	19	15 24	12		150	
16 r w		15	57	15	55 62	41 48	28 35	12	75	
17 r w		4	16	2	12 27	9 17	6 9	4 9	300	
18										
19 r w			3	13	2	19 19	13 16	7 13	8	150
20 r w			6	15	5	15 22	12 18	8 14	9	150

r = at rest
w = at work

(PREOPERATIVE)

Case No.	Blood Pressure mm Hg							Work load Kgm/min	
	Venous	R A M	R V		P A				PCV M
			Syst.	End-diast.	Syst.	M	Diast.		
21	r w	12	40	22	36 38	25 31	20 25	16	150
22	r w	16	50	16	52 66	41 50	27 24	13	75
23	r w	27	29	15	23 36	17 28	11 22		75
24	r w	16	45	28	56 48	37 37	24 27		75
25	r w	8	33	8	26 39	21 30	13 25	15	150
26	r w	8	31	10	36 39	26 29	19 24	17	70
27	r	6	52	7	32	19	9	10	
28	r w	14	27	16	30 35	24 30	19 24	16	75
29	r	14	29	11	27	19	13	13	
30	r w	18	31	11	34 44	28 37	23 31	18	75
31	r w	19	36	15	39 47	29 34	24 23	23	145
32	r w	25	64	22	60	43 72	32	19	80
33	r w	13	26	16		24 26		19	200
34	r	7	27	9	31	24	17	21	
35	r w	10	51	10	50	29 44	21	12	90
36	r w	14	49	17	42 83	34 65	25 51	26	150
37	r	14	33	13		22		15	
38	r	17	22 (M)			24			

r = at rest
w = at work

(POSTOPERATIVE)

Case No.	Blood Pressure mm Hg								Work load Kg _m /min
	Venous	R A M	R V		P A			PCV M	
			Syst.	End-diast.	Syst.	M	Diast.		
21 r w		3	17	4	20 18	14 14	9 9	1	150
22 r w		7	31	3	31 57	24 43	14 23	9	150
23									
24									
25 r w		5			22 41	14 26	6 15		70
26 r w		4	32	4	40 55	34 42	21 28		150
27 r r		4.5 6	24	6	17	11	5	6	
28 r w		2	21	3	23 21	14 17	8 12	4	150
29 r w		—1	16	1	13 14	8 10	3 4	5	150
30 r w		3			20 28	12 18	4 6	5	150
31									
32 r		5	33	6	33	23	15		150
33 r w		—1	30	3	23 26	13 17	4 7	3	150
34 r w		—2	25	0	24 19	15 15	6 6	6	150
35									
36 r		6	50	9	33	25	18		

r = at rest

w = at rest

Preoperative				Postoperative		
Case No.	V C	$\frac{VC \times 100}{PVC}$	$\frac{RC \times 100}{TC}$	VC	$\frac{VC \times 100}{PVC}$	$\frac{RC \times 100}{TC}$
1				4.53/5.10	88.8	35.2
2						
3						
4						
5	3.38/3.71	91.0		3.52/5.3	66.4	34.6
6	2.16/3.65	59.2		3.23/4.6	70.2	33.7
7	3.5/3.86	90.5		3.72/4.7	79.1	26.0
8	2.73/3.92	69.6				
9	3.15/4.9	64.3	19.6	5.28/5.1	103.0	21.1
10	5.01/4.9	102.0	21.4	5.10/5.10	100.0	22.1
11				3.92/5.30	73.9	27.5
12	1.67/4.5	37.0	28.9	2.06/4.4	45.8	33.0
13	3.92/5.6	70.0	27.5	5.16/5.5	93.8	29.5
14				0.77		38.9
15				2.88/6.0	48.0	33.8
16				2.77/3.5	79.1	27.9
17	4.53/4.9	92.5	21.2	3.62/4.9	73.9	22.0
18	2.4/4.1	58.5				
19						
20	4.36/5.1	85.5	16.5	4.53/5.1	88.8	15.3
21				4.93/4.5	109.0	26.3
22	2.24/3.6	62.1	33.0	2.03/3.6	56.4	30.0
23	1.3/2.85	45.5				
24	0.76/2.74	27.8				
25	2.92/4.9	59.5	38.1	2.58/4.9	52.7	38.4
26	2.66/5.0	52.0	26.3	2.64/5.0	52.8	30.0
27	3.62/5.5	65.7	39.0	2.61/5.5	47.5	46.0
28	2.53/5.0	50.6	28.7	3.93/5.2	75.6	21.2
29				3.48/5.2	66.9	13.4
30	2.13/4.4	48.5	21.0	3.09/4.6	67.2	36.8
31	1.87/4.2	44.5	38.7	3.01/4.0	75.3	24.2
32	1.21/4.7	25.7	14.2	2.02/4.7	43.0	36.5
33	5.48/5.7	96.0	25.3			
34	3.3/5.5	60.0	23.8	4.7/5.6	83.9	16.4
35	2.5/4.6	54.4	32.4			
36	2.68/5.5	48.7	28.0	5.14/5.4	95.2	28.9
37						
38						
M $\frac{VC \times 100}{PVC} = 62.3 \pm 4.2$ (25 cases)				M $\frac{VC \times 100}{PVC} = 73.1 \pm 3.7$ (25 cases)		
M $\frac{VC \times 100}{PVC} = 63.5 \pm 4.8$ (19 cases)				M $\frac{VC \times 100}{PVC} = 72.1 \pm 5$ (19 cases)		

Vital capacity = VC
 Predicted VC = PVC
 Residual capacity = RC
 Total capacity = TC

Preoperative							Postoperative					
Case No.	Serum protein %	Albumin %	Globulin %	Thymol turbidity units	Alkaline phosphatase units/100 ml	Serum bilirubin mg/100 ml	Serum protein %	Albumin %	Globulin %	Thymol turbidity units	Alkaline phosphatase units/100 ml	Serum bilirubin mg/100 ml
1	7.8	5.1	2.6	Takata	13.3	1.4	6.2			0.2	6.8	0.8
2				Takata	13.0	1.2						
3						6.4						
4	3.06	1.73	1.17	Takata —	8.9	0.6	6.4	3.8	2.6	1.0	7.7	0.23
5	7.5			Takata —	11.7	0.58	6.6			2.7	8.1	1.17
6	7.0			Takata +	9.6	0.55	7.4				8.9	0.88
7	6.9	5.0	1.9	Takata +	6.2	0.90	7.0			1.2	6.8	1.67
8	7.1	4.8	2.5	Takata —	18.2	2.61						
9	5.29	3.41	1.88			0.72	7.3					0.92
10	6.8			0.5	6.5	1.8	6.64	4.96	1.68	1.0	8.9	0.31
11	6.6			2.4		0.6	5.89	3.85	2.04	1.6		
12	6.83	4.0	2.83		11.4	0.68	7.0			1.1	5.0	1.10
13	5.8	4.28	2.46		8.1	2.81	6.4			0.3	5.5	1.26
14	5.2	3.0	2.2	2.9	8.5	0.25	6.2	4.3	2.2			
15	4.1	2.6	1.5	1.6	3.6		6.8	4.9	1.95	10.7	6.7	0.64
16	7.0			2.0	6.0	1.3	7.7	4.2	3.5	2.0	5.0	1.2
17	7.12	3.44	3.68			1.08	6.4			1.0	5.3	
18	5.68	3.78	1.90	2.8	23.2	0.89						
19	6.5	3.9	2.6		16.0	3.0	8.2			2.0	17.7	0.69
20	9.26	3.64	5.62	1.0	7.0	0.6	5.12	3.76	1.36	2.0	5.9	
21	< 4.9	2.73	2.18	0.5	7.6	0.51	6.12	4.75	1.37	3.7	6.8	0.33
22	7.6	4.3	3.3	3.0	16.0	2.36	7.5	4.63	2.87	1.0		0.6
23	6.0			6.2	14.3	0.56						
24	6.1			1.2	8.1							
25	7.54	5.15	2.39	2.7	8.6	1.82	6.6			3.6	6.8	2.25
26	5.0			0	14.0	0.41	6.51	5.29	1.22	1.0	9.9	0.75
27	7.1	5.56	1.54	1.6	8.8	0.51	7.6	4.35	3.25	4.0	7.0	0.6
28	6.6			0.1	14.0	0.8	6.6			1.5	7.4	0.64
29	7.6	3.1	4.5	9.9	12.6	1.34	8.0	5.02	2.98	7.1		0.7
30	6.2	3.86	2.34	2.0	15.0	0.2	6.69	4.74	1.95	0.5	5.6	0.54
31	7.84	5.55	2.29	2.0	6.0	0.69						
32	7.2			6.2	17.0	0.51	6.4			2.8	4.6	1.03
33	6.2			1.5	3.8	0.98	6.31	4.74	1.57	2.5	4.4	0.69
34	4.0			3.2	14.0	0.59	7.08	4.79	2.29	6.0	8.7	0.55
35	6.1	3.2	2.9	4.6	19.0	0.92						
36	7.1	4.2	2.9	2.7	17.0	1.09	5.8	4.15	1.65	2.0	7.2	0.38
37	6.4	4.1	2.3	1.0		2.35						
38	7.5	4.2	3.3	4.6	1.0							

Preoperative											Postoperative										
Case No.	Chronic, ill	Venous stasis	Cyanosis	Dyspnoea at rest	Oedema	Liver enlargement	Ascites	Blood pressure Syst./Diast. mm Hg	Rhythm	Protodiast. triple rhythm	Pleural exudate		Chronic, ill	Venous stasis	Cyanosis	Liver enlargement	Blood pressure Syst./Diast. mm Hg	Rhythm	Protodiast. triple rhythm		
											Right	Left									
1	—	+	—	—	—	+	—	160/100	—	+	—	—	—	—	—	—	150/85	—	—		—
2	+	+	+	—	—	+	+	100/60	AF	—	+	+	—	—	—	—	130/70	—	—		—
3	+	+	+	—	++	+	+	110/75	—	+	+	+	—	—	—	—	135/80	—	—		—
4	+	+	+	—	++	+	+	115/80	—	+	+	+	—	—	—	—	130/80	AF	—		—
5	—	—	—	—	—	+	+	115/85	AF	—	—	—	+	+	+	+	120/60	AF	—		—
6	+	+	+	—	+	+	+	120/90	AF	+	—	+	—	—	—	—	125/85	AF	—		—
7	—	—	—	—	—	+	—	115/80	AF	+	—	—	—	—	—	—					
8	+	—	—	+	?	+	+	140/90	AF	+	+	+	—	—	—	+					
9	+	+	+	—	—	+	(+)	120/80	—	—	—	+	—	—	—	—	135/70	—	—		—
10	—	+	+	—	—	+	+	130/105	—	—	—	+	—	—	—	—	125/75	—	—		—
11	—	+	—	—	—	+	+	110/80	—	—	—	—	—	—	—	—	120/60	—	—		—
12	+	+	+	—	+	+	+	115/80	—	+	+	+	—	—	+	+	120/70	—	+		—
13	—	—	—	—	—	+	—	135/100	—	+	+	+	—	—	—	—	125/75	—	—		—
14	+	+	+	+	+	++	+	105/85	—	—	+	+	—	—	—	—	125/80	—	—		—
15	—	+	+	—	+	+	+	110/85	—	—	+	+	—	—	—	—	135/75	—	—		—
16	—	+	+	+	+	+	—	175/120	AF	—	+	+	—	—	+	—	145/95	AF	—		—
17	—	+	—	—	—	(+)	+	105/80	—	+	—	—	—	—	—	—	135/75	—	—		—
18	+	+	+	—	—	+	+	160/110	—	—	+	+	—	—	—	—					
19	+	+	—	—	?	+	—	105/60	?	+	—	—	—	—	—	—	140/90	—	—		—
20	+	—	—	—	—	+	++	130/105	AF	+	—	—	—	—	—	—	115/70	AF	—		—
21	—	—	—	—	—	+	+	130/80	—	+	—	+	—	—	—	—	125/75	—	—		—
22	+	+	+	—	+	+	(+)	135/95	—	+	—	—	—	—	—	—	140/85	AF	—		—
23	+	+	+	—	+	+	+	100/70	—	—	+	+	—	—	—	—					
24	+	+	+	—	—	+	—	110/80	AF	—	+	+	—	—	—	—					
25	—	—	—	—	—	+	—	150/95	AF	—	—	—	—	—	—	+	150/95	AF	—		—
26	—	+	—	—	+	—	—	140/100	AF	—	—	—	—	—	—	—	135/80	AF	—		—
27	—	+	+	+	—	+	—	130/80	—	+	—	—	—	—	—	++	130/75	—	—		—
28	—	—	+	—	—	+	+	125/90	—	—	+	+	—	—	—	—	145/80	—	—		—
29	—	—	—	—	—	+	—	125/90	—	+	+	+	—	—	—	—	135/85	—	—		—
30	+	+	+	—	+	+	—	95/70	—	—	+	+	—	—	—	—	110/75	—	—		—
31	—	+	—	—	+	+	—	130/85	—	+	—	—	—	—	—	—	135/75	—	—		—
32	+	+	+	+	—	+	+	120/80	AF	—	—	—	—	—	—	—	110/65	AF	—		—
33	—	—	—	—	—	—	—	115/70	—	—	—	—	—	—	—	—	125/85	—	—		—
34	—	+	—	—	+	++	—	150/80	—	—	—	—	—	—	—	—	120/75	—	—		—
35	—	+	—	—	—	+	—	105/80	AF	+	—	—	—	—	—	—					
36	—	+	—	—	—	++	—	130/90	AF	—	—	—	—	—	—	—	135/75	—	—		—
37	+	—	—	—	—	+	—	125/90	—	—	—	—	—	—	—	—					
38	—	+	+	—	—	++	—	120/105	—	—	+	—	—	—	—	—					

AF=atrial fibrillation

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